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### THE MANAGEMENT OF THE PATIENT WITH A HAEMOSTATIC DEFECT DURING THE PERIOD OF SURGERY.<sup>1</sup>

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TWO PROBLEMS may confront the surgeon when it is desirable to have information about a patient's haemostatic mechanism. The more common is that of the person admitted to hospital for elective surgery who volunteers the information that he is a "bleeder". The second is that of the known bleeder in whom surgical intervention is necessary, perhaps as a life-saving measure. Both problems can be solved only if laboratory facilities are adequate to investigate haemorrhagic disorders.

The investigation of the patient who may be a bleeder must include a careful clinical history and sufficient laboratory tests to exclude the known causes of abnormal bleeding. The assessment of a patient's haemostatic mechanism is essentially a process of exclusion, although certain points in the history may be valuable. A family history of abnormal bleeding should always suggest the need for further investigation. A history of abnormal

bleeding after tooth extraction is particularly important. Bleeding after tooth extraction which persists for 48 hours, or which requires a blood transfusion, almost certainly indicates defective haemostasis.

The known bleeder who must undergo surgery cannot be intelligently managed unless the nature of the haemostatic defect is clearly understood. Laboratory facilities must also be adequate to control therapy during the period when the haemostatic mechanism is under stress as a result of the surgical procedure.

It is therefore worthwhile considering the minimum laboratory tests necessary to investigate a possible bleeder. It is a popular misconception that estimation of the bleeding time and the whole-blood coagulation time are effective screening tests. It is advisable to look critically at the information obtained from these tests. The bleeding time is said to be prolonged in thrombocytopenic purpura, and also in a mixed group of bleeding disorders thought to be due primarily to a capillary vascular abnormality and collectively known as von Willebrand's disease. The bleeding time is normal in most haemorrhagic disorders due to deficiency of a blood-clotting factor. Its estimation is a most inaccurate test, depending more than is thought on the exuberance with which the skin is punctured. The normal range is by no means certain, and repeated tests on the same subject can give a wide range of values. In von Willebrand's disease, the bleeding time fluctuates from day to day and may be normal at the time of testing.

If the whole-blood coagulation time is prolonged, it is certain that the patient suffers from a coagulation defect.

<sup>1</sup> Read at a meeting of the Section of Pathology, Australasian Medical Congress (B.M.A.), Tenth Session, Hobart, March 1 to 7, 1958.

The inference cannot be drawn that a normal coagulation time excludes such a defect. The classical disease associated with a prolonged coagulation time is haemophilia. Even in this disease, the coagulation time may be normal. In a series of 65 consecutive patients with haemophilia whom I examined, the coagulation time was normal in 19. Similar observations have been made previously by others. Haemophilia associated with a normal coagulation time is usually mild from the viewpoint of spontaneous symptoms; but such patients often bleed excessively when the haemostatic mechanism is put under stress, as after tooth extraction or a surgical operation. In coagulation defects due to abnormalities of the prothrombin complex, a normal clotting time is the rule.

The number of clotting factors in normal blood is not certain, and the list is constantly being amended. The series of interactions which take place during blood coagulation are very imperfectly understood; but a working theory is essential, not only to plan the laboratory investigation of a haemostatic defect, but to manage intelligently the patient who is a bleeder. Such a working theory is illustrated in Figure 1. The most recent

#### A theory of in-vitro blood coagulation:

1. Antihæmophilic globulin (AHG) — Christmas factor  
Factor V  
Factor VII  
Factor X  
Platelets  
Calcium
2. Plasma thromboplastin + calcium + prothrombin → thrombin
3. Thrombin + fibrinogen → fibrin

FIGURE 1.

advances in knowledge concern the thromboplastin-precursor substances and the nature of plasma thromboplastin. The more important hæmorrhagic disorders are associated with deficient production of plasma thromboplastin. A laboratory test—the thromboplastin generation test—is now available which detects the ability of a patient's blood to manufacture plasma thromboplastin. The results of this test give an indirect evaluation of the concentration of thromboplastin-precursor substances. In the investigation of any patient, especially a male, who may have a coagulation defect, the investigation is not complete until the thromboplastin generation test has been performed. This is because the thromboplastin generation test may be the only abnormal finding in the investigation of certain bleeding states, especially mild haemophilia. Obviously, screening tests to detect abnormal bleeding states must be the most sensitive available.

In our laboratory we use the following tests as a screening procedure for any patient whose history suggests that he may be a bleeder: Hess (tourniquet) test; examination of a blood film; platelet count; estimation of bleeding time; one-stage estimation of prothrombin time; thromboplastin generation test. If an abnormality is detected with any of these tests, further lines of investigation are followed. A simplified screening thromboplastin generation test has been published which obviates the necessity to carry out the time-consuming procedures of treating plasma with aluminium hydroxide and washing platelets. By the use of this screening method, all the tests listed above can be carried out in most hospital laboratories, and the whole series of tests should not take more than one hour.

The management of the patient with a bleeding tendency for whom surgery is indicated is modified by the nature of the defect present. In this paper, the more common bleeding disorders will be considered, and an account given of some personal experiences with such patients.

#### Thrombocytopenic Purpura.

Splenectomy is still the treatment of choice for certain patients with thrombocytopenic purpura. The factors which influence the choice of patient for splenectomy are

outside the scope of this paper. Oozing of blood from the wound or splenic bed in patients with thrombocytopenia may create a problem in surgical management. This complication can be minimized by intelligent pre-operative management. It is generally accepted that operative bleeding is less in patients with thrombocytopenia who are receiving steroid therapy. The steroid drugs, especially cortisone and prednisolone, are undoubtedly useful in the medical treatment of thrombocytopenia. Their mechanism of action is to improve capillary resistance without necessarily raising the platelet count. If a patient with idiopathic thrombocytopenic purpura is given adequate steroid therapy, it is usual to find that the response to the tourniquet test becomes negative, the bleeding time becomes normal, and spontaneous bruising and purpura cease within three to five days. There is usually no change in the platelet count. If such a patient is submitted to splenectomy, steroid therapy should be continued. One reason is that there are added anaesthetic risks associated with surgical procedures in cases in which the steroids have been recently withdrawn. For the patient with chronic idiopathic thrombocytopenic purpura with mild symptoms in whom spontaneous purpura is not a feature, pre-operative steroid therapy may not be warranted. Six patients with idiopathic thrombocytopenic purpura underwent splenectomy at the Royal Perth Hospital in the past year. Four of these had severe symptoms and were treated pre-operatively with cortisone or prednisolone. The remaining two did not have severe symptoms and were operated on without steroid cover.

Transfusion of platelet-rich fresh blood at the time of operation is a useful preventive measure for operative and post-operative oozing from the wound and splenic bed. This is despite the fact that a circulating platelet antibody is present in many patients with thrombocytopenic purpura, and the survival time of transfused platelets is very short. Intact platelets are not essential for hæmostasis. It has recently been shown that frozen platelet suspensions exert a hæmostatic function similar to that of intact platelets. This function possibly is due to the ethanolamine phosphatide content of the platelets.

Platelet-rich blood may be obtained by taking blood from donors through plastic taking sets into plastic transfusion bags. The blood is administered through a plastic giving set. Blood drawn on the day of operation should be used. We have found that, even after 24 hours in such a bag, blood collected through the giving set contained nearly the same concentration of platelets as blood sampled directly from the donor. The platelet morphology as observed on stained films showed little change from normal.

#### Hæmophilia.

The coagulation defect in hæmophilia is caused by a deficiency or complete absence of plasma anti-hæmophilic globulin (AHG). AHG is an essential thromboplastin-precursor substance. Deficiency causes defective plasma thromboplastin production and imperfect conversion of prothrombin to thrombin during *in vitro* coagulation. Hæmophilia can be detected in the laboratory by the thromboplastin generation test or by one of several methods of AHG assay. Bleeding is likely to occur when the plasma AHG concentration is less than 30% of normal. This is about the level at which the thromboplastin generation test will give an abnormal result. It is important to emphasize that the whole-blood coagulation time in hæmophilia can be expected to be normal at AHG concentrations above 2%.

If the plasma AHG concentration can be kept about 30% of normal during the period of operation and for a few days afterwards, even major operations can be carried out on hæmophiliacs without excessive bleeding. The most readily available source of AHG is fresh normal plasma. Serum is useless in the treatment of hæmophilic bleeding. The AHG concentration of plasma stored at 4° C. rapidly decreases, and 50% may be lost in 24 hours. To be most effective, plasma should be separated within two hours of collection of the blood and used immediately. Plasma may be stored frozen at -20° C. with only small loss of potency if it is separated and frozen immediately after collection of the blood.



The general tendency has been to give too little plasma in an endeavour to correct the abnormal haemostatic mechanism in haemophilia. Some theoretical considerations should make this plain. If a haemophilic has a zero plasma concentration of AHG, a third of his plasma volume will have to be fresh normal plasma to give a concentration of about 30%. On theoretical grounds, this would entail transfusing a litre of fresh plasma. However, not all the transfused AHG can be accounted for in the circulation. Some may escape into the tissues; it is also probable that an inhibitor of AHG occurs in the plasma of many haemophiliacs. A priming dose of considerably more than a litre of fresh plasma is often necessary to achieve a plasma AHG concentration of 30%. The main difficulty associated with maintaining this plasma level of AHG is the rapid turnover in the recipient's plasma. Injected AHG has a half-life of a few hours only in the circulation of a haemophilic.

Maintenance therapy must be continuous, and sufficient to ensure a normal haemostatic mechanism perhaps for several days. A rough working principle is to transfuse one litre of fresh plasma immediately before surgery as fast as possible, and then maintain the plasma drip administration at the rate of four millilitres per kilogram of body weight every four hours. An adult usually requires two and one-half litres of fresh plasma in the first 24 hours, and then one and one-half to two litres a day afterwards. The minimum effective control of therapy is the thromboplastin generation test, the result of which should remain normal. It is important that the actual surgery be carried out at the time when the patient's haemostatic mechanism is effective. To operate on a haemophilic without prior therapy in the vain hope that he will not bleed, and then to attempt a delayed correction of the haemostatic mechanism, is to lose the great benefit of effective primary coagulation in severed vessels. It is much easier to prevent a haemophilic from bleeding than it is to stop bleeding once it is uncontrolled.

Over the past two years, we have used these principles of replacement plasma therapy in four cases of haemophilia with satisfactory results. An illustrative case is as follows.

A man, aged 26 years, has moderately severe haemophilia. He has a "zero" plasma concentration of AHG. He suffers from spontaneous bruises and haemarthroses. Haemorrhage continued for three weeks after tooth extraction, and he required transfusions of 14 pints of blood. He suffers periodically from haematuria. He was admitted to hospital on January 23, 1957, with a history that five days previously he had been involved in a motor-car accident. He noticed haematuria shortly afterwards, but did not report for treatment until he suddenly collapsed with acute abdominal pain and signs of acute blood loss. Examination disclosed signs of intraperitoneal and retroperitoneal haemorrhage. Rupture of the spleen was thought a likely diagnosis, but because of the complicating retroperitoneal haemorrhage and the knowledge that he was a haemophilic, he was treated expectantly at first. He was given transfusions of large volumes of fresh plasma and fresh blood. The result of the thromboplastin generation test became normal, and a plasma AHG concentration between 28% and 50% was maintained. As bleeding continued into the peritoneal cavity despite the correction of the haemostatic defect, a definite pre-operative diagnosis of rupture of the spleen was made. Four days after his admission to hospital, after intensive pre-operative plasma therapy which raised the plasma AHG concentration to over 50%, the peritoneal cavity was cleared of blood and a ruptured spleen removed. Plasma therapy was continued for a further four days, and the result of the thromboplastin generation test remained normal or almost so. There was no post-operative bleeding, and except for a small haematoma about the drainage tube, the operation wound healed by first intention. The patient's haemophilic state has not been affected by the splenectomy.

Recent intensive work in England has made available a concentrated preparation of AHG produced from ox blood. When reconstituted for injection, this material has, volume for volume, about 80 times the antihemophilic potency of fresh human plasma. The advantages of this material are that much smaller volumes need to be injected, which reduces the danger of circulatory overload in seriously ill patients, and that in haemophiliacs much higher plasma

AHG concentrations can be obtained than is possible with human plasma. The material is antigenic, and loses its effect after continuous treatment for several days. Therapy may also be associated with severe reactions. Nevertheless, bovine AHG has been used with considerable success in England for surgical procedures on haemophilic patients. Recently we had the opportunity to use bovine AHG supplied by Dr. R. G. Macfarlane in the care of a severely haemophilic patient in whom major surgery was necessary.

A severely haemophilic patient, aged 21 years, developed an extensive blood cyst of the thigh, which slowly pointed to the surface and discharged old blood. After several days the blood cyst became infected with an antibiotic-resistant *Staphylococcus aureus*. The patient developed extensive cellulitis of the thigh, with grave toxic signs. Immediately after an injection of 60 cubic centimetres of bovine AHG (with a plasma equivalent of nearly five litres), an extensive incision was made in the thigh, much infected blood clot removed, the operation area debrided of necrotic muscle and the wound closed loosely with drainage. Injections of bovine AHG were given daily or twice daily thereafter, therapy being controlled by plasma AHG assay. Plasma AHG concentrations were kept between 27% and 60% of normal until the sixth post-operative day, when evidence of refractoriness developed (Figure II). Bovine AHG injections were continued for a further four days, but had little effect on the plasma AHG concentration. There was persistent mild oozing of blood from raw surfaces, but never haemorrhage severe enough to cause alarm. The material undoubtedly was effective in controlling bleeding. Previous experience had shown that this patient was refractory to human plasma therapy.

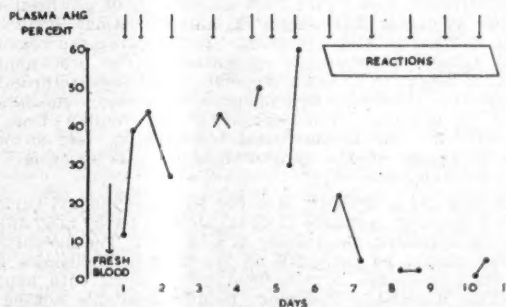


FIGURE II.

Plasma AHG concentrations in a severely haemophilic patient treated by injections of AHG concentrate of bovine origin.

#### Von Willebrand's Disease.

This imperfectly understood group of bleeding disorders comprises those encountered most commonly in practice. The essential defect is thought to be a capillary abnormality inherited in autosomal dominant fashion. The capillaries have been reported as morphologically abnormal when viewed under the skin microscope or with the slit lamp. They do not contract normally when injured. The most important laboratory findings are a positive response to the tourniquet test and a prolonged bleeding time. The platelets and coagulation factors were thought to be normal until recently; at least some of these patients have a reduced concentration of plasma AHG. At times the bleeding time and the result of the tourniquet test may be normal and diagnosis can be made then only on the history. A limited experience with patients in this group suggests that elective surgery can be carried out successfully if it is performed when the bleeding time is normal and the response to the tourniquet test negative. One patient has had several abdominal operations performed without excessive bleeding at times when the laboratory tests gave normal results. The bleeding time in this patient fluctuates greatly from day to day. The procedure has been to admit her to hospital and carry out daily tests of bleeding time and capillary fragility. As soon as normal values are obtained, surgery is carried out without any other precaution. Another patient recently under-

went hemorrhoidectomy without excessive bleeding at a time when the bleeding time was normal. There are conflicting reports concerning the value of steroid therapy in preventing operative hemorrhage in von Willebrand's disease. The condition is so liable to spontaneous fluctuations in severity that controlled evaluation of any therapy is difficult. Cortisone treatment has been given to three patients over the past two years. Two of these had teeth extracted without excessive bleeding; the third underwent a normal confinement without incident. The procedure has been to give the patients 200 milligrammes of cortisone daily for two days before and two days after the operative incident.

In patients with von Willebrand's disease who have low plasma AHG concentrations, intensive therapy with fresh plasma before surgery would appear to be rational.

#### Liver Disease.

In considering the hemostatic defect in liver disease, distinction should be made between obstructive jaundice and hepatocellular damage. The patient with obstructive jaundice is likely to bleed because the body suffers from a deficiency of vitamin K. This cannot be absorbed from the intestinal tract. If vitamin K is administered parenterally, the coagulation defect is corrected and surgery can be performed without danger of hemorrhage.

The patient with hepatocellular damage does not suffer from vitamin K deficiency, and vitamin K therapy is ineffective in correcting any hemostatic defect that may be present. It is probable that the liver is responsible for the manufacture of many coagulation factors. Patients with cirrhosis may suffer from a deficiency of prothrombin, factor V, factor VII, factor X and Christmas factor as well as from thrombocytopenia. In assessing the coagulation defect, the one-stage estimation of the prothrombin time is useful to detect abnormalities of the prothrombin complex. However, thromboplastin-precursor substances may be deficient even when the prothrombin time is normal, and the thromboplastin generation test as well as estimation of the prothrombin time is essential for proper evaluation.

In treating a cirrhotic bleeding from oesophageal varices, it is important to realize that, in addition to the mechanical factors operating, the patient is a bleeder. The hemostatic defect should be corrected as far as possible before any surgical procedure is performed on a patient with hepatocellular damage. The most readily available source of coagulation factors is fresh human plasma, and adequate plasma therapy is a rational way to correct the bleeding tendency. Therapy should be controlled by both the prothrombin time estimation and the thromboplastin generation test.

A female, aged 35 years, suffered from chronic hepatitis with splenomegaly and ascites. The prothrombin time was 19 seconds (normal control 12.5 seconds) and was unaffected by vitamin K therapy. The result of the thromboplastin generation test was abnormal. Laparotomy was decided on to evaluate the possibility of a shunting operation. Immediately before surgery, one litre of fresh plasma was transfused. The prothrombin time was still abnormal after this (16 seconds), but the result of the thromboplastin generation test was now normal. Laparotomy was performed without excessive bleeding, and the wound healed without hematoma formation. Radical surgery was not possible.

#### Conclusions.

The major part of this paper has been devoted to emphasising the importance of a planned programme when surgery has to be carried out on a patient who is a bleeder. The nature of the coagulation defect must be clearly understood, and facilities available for adequate laboratory control during the period of surgery. Although only four hemorrhagic disorders were chosen to illustrate the problems involved, similar principles apply to any type of coagulation defect. It has been possible to consider the importance of systemic therapy only in controlling bleeding during surgery. However, a careful surgical technique and the application of local hemostatic measures are essential components of the programme. Various clotting agents, such as thrombin solution, fibrin

foam and viper venom, may have a place in therapy as local agents. The hematologist is often in a position to advise the surgeon which agent to use in a particular circumstance.

With increasing knowledge of the hemorrhagic disorders and better techniques for refining and concentrating the coagulation factors from human blood, we can look forward to an era when the bleeder will be subjected to only slightly greater risk than the normal person at operation, and elective surgery can be carried out with confidence.

#### Summary.

Major surgery is possible in patients with a coagulation defect provided that the nature of the bleeding tendency is clearly understood and that the defect is temporarily correctable. Surgery should not be undertaken until the defect has been corrected as far as is possible. Adequate laboratory facilities for the diagnosis and control of hemorrhagic disorders are essential if surgery is to be successful.

The principles of management in four common groups of hemorrhagic disorders are discussed with reference to illustrative case reports.

#### Acknowledgements.

I wish to thank Dr. R. G. Macfarlane, Radcliffe Infirmary, Oxford, England, for a supply of AHG concentrate of bovine origin. It is a pleasure to acknowledge the cooperation of the honorary surgeons of the Royal Perth Hospital.

#### MEDICAL SECRECY IN THE LAW<sup>1</sup>

By D. P. O'CONNELL, LL.D.,  
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Adelaide.

It is widely believed that the medical practitioner, being in the nature of a confidant and an adviser, has some privilege in the matter of silence concerning his client's disclosures and his own discoveries. However, there is in fact no privilege whatever attaching to the practitioner-client relationship, and indeed there is no reason why there should be. To take one example, a spouse in a paternity or affiliation suit might be hard put to establish anything at all if the evidence of medical examination of the opposite spouse were not compellable in court. At least since the famous case of the Duchess of Kingston in 1776, it has been the rule that, whatever medical etiquette may require, a medical practitioner is obliged if called upon to give evidence in a court of law. He may be asked to disclose information which came to him through his professional relationship with his patient. It has been held, for example, that a medical practitioner who in the course of treating a patient under a national health scheme ascertains that the patient is suffering from venereal disease, may be compelled to give evidence to that effect, even though the statutory regulations applying to the scheme enjoin absolute secrecy.

Far from being privileged to remain silent, and contrary to what is popularly believed, and even to what is probably medical ethics, a doctor is bound to communicate with the police if his client discloses the commission of a criminal offence. So it was held in 1914 by a judge in his charge to a grand jury in Birmingham.

The disclosures just mentioned are, of course, disclosures to a court or to the police. The case of disclosures out of court is naturally quite different. This is not a question of the law of evidence relating to the proof of facts, but a question of the responsibility which the practitioner owes to his client not to broadcast his or her medical condition. English law does not impose upon any person an obligation of silence. A newspaper, for example, can publish a photograph of a distinguished personality in an embarrassing situation; but provided the publication does

<sup>1</sup> Read at a meeting of the South Australian Branch of the British Medical Association.



not amount to libel, there is little that can be done about it. There is very little law on the question of invasion of privacy in circumstances not amounting to libel.

The doctor is in no fundamentally different position from that of the newspaper. He is restrained by the law of defamation and by the law of negligence. Let us take each of these in turn.

The law of defamation subdivides into libel, which is a written statement, and slander, which is spoken. In both cases the statement is defamatory if made in circumstances which bring the person commented upon into hatred, contempt or ridicule, and which cause him to be shunned and avoided. In the other States of Australia the truth of the statement is a defence only if there was some public benefit in its disclosure. This means that a true statement by a medical practitioner, made either in writing or by word of mouth, that his client was suffering from venereal disease would be defamatory, at least if made in ordinary conversation. In South Australia, however, the theory is that there is no defamation if the person defamed has his character reduced only to its proper level. Hence, in the case mentioned, an indiscreet doctor would not be liable in law for his disclosures. Since it is unlikely that doctors will tell deliberate falsehoods, this topic may be left at this point, and consideration directed to negligence as the only legal restraint on medical loquacity.

Before we do this, however, it may be well to examine the question whether or not a doctor by implication contracts with his client to remain silent. The question does not appear ever to have come before a court. On general principles, I would suggest that a term to the effect that the relationship will be confidential could be implied in the client-practitioner relationship. Since, however, the only damages that can arise in contract are those which are actually suffered in the way of specific loss, and not general damages for mental pain and suffering, it would in most instances not be profitable for a client to bring an action.

A client harmed by disclosures must, in order to recover damages, it seems, found his action in negligence. It is, of course, obvious that statements made carelessly by a doctor which prove to be not strictly accurate are negligent statements; but it perhaps comes as a surprise to be told that statements made which are true, and which are made with the most serious intent and after great deliberation, may also be negligent. It must be realized that the word "negligence" in English law has in the past 30 years lost its traditional association with recklessness, and come to refer to a general principle or theory of duty which may be substantially independent of what the man in the street considers negligence.

This leads me to a discussion of the extraordinary case of *Furniss v. Fitchett*. It should be mentioned that this is the first decision in English law in which a doctor has been held liable to his patient for disclosure, and especially since it is not a Court of Appeal decision it may not necessarily be followed elsewhere. It is, however, a useful guide to us in our consideration of the general problem of medical secrecy.

The facts were as follows. Dr. Fitchett (the defendant) was and had been the regular medical attendant of both Mrs. Furniss (the plaintiff) and her husband. It appears that Mrs. Furniss entertained suspicions that her husband was "doping" her and that he was insane. She told her doctor that her husband was cruel to her and occasionally even violent. These suspicions and charges were quite without foundation; but they naturally engendered a certain amount of domestic discord, which in turn affected the health of the husband. A separation of husband and wife, temporarily at all events, would probably have been in the interest of the health of both of them, and a separation had been discussed but not arranged. Dr. Fitchett had been asked by Mr. Furniss's solicitor whether he could arrange for Mrs. Furniss to be "certified". Had she been committed to a mental institution, that would, of course, have brought about at least a temporary separation, and would have relieved Mr. Furniss from the anxieties which were adversely affecting his health. Dr.

Fitchett said in evidence that he was "deeply worried over the suggestion of certification". Though he thought that Mrs. Furniss showed symptoms of paranoia, he did not think that to commit her to a mental institution would be in her best interest. As family doctor he was faced with a problem that presented many difficulties. He was attempting to give medical advice to two persons—husband and wife—whose interests, even if they were not, from a medical point of view, really in conflict, may well have seemed to his patients to be in conflict. That was the situation which existed on May 21, 1956.

On that date Mr. Furniss saw the defendant. The defendant's evidence was that his patient was then almost desperate, that he was in a distraught state, and that he said: "You must do something for me doctor—give me a report for my lawyer." After deep thought, the defendant then wrote out, signed and gave to Mr. Furniss a document which was worded as follows:

21.5.56.

Mrs. Phyllis C. L. Furniss,  
32 Mornington Road.

The above has been attending for some time and during this period I have observed several things:

- (1) Deluded that her husband is doping her.
- (2) Accuses her husband of cruelty and even occasional violence.
- (3) Considers her husband to be insane and states that it is a family failing.

On the basis of the above I consider she exhibits symptoms of paranoid and should be given treatment for same if possible. An examination by a Psychiatrist would be needed to fully diagnose her case and its requirements.

Yours faithfully,  
A. J. FITCHETT.

The evidence does not reveal what was immediately done with this document. Mrs. Furniss continued to see the defendant professionally down to the month of April, 1957. From June 3, 1956, to August 7, 1956, Mrs. Furniss was away from her home. She then returned for a short period, but left again on October 4, 1956. Later she took proceedings against her husband for separation and maintenance orders. The application was heard in the Magistrates' Court on May 29, 1957. During the hearing and in the course of cross-examination of Mrs. Furniss, Mr. Furniss's solicitor produced to her the above-quoted document of May 21, 1956. It was a little more than a year old; but it was only then that Mrs. Furniss learned of its existence.

Mrs. Furniss brought an action before a judge and jury. The latter was asked whether the doctor owed a duty to his patient, and if so, whether it had been violated. The jury returned a verdict that there was a duty and awarded damages. The defendant then moved the court that there was no cause of action. In effect this meant that the court was asked to hold that the plaintiff in law had no case to put to the jury. The Chief Justice recognized the complete novelty of the claim. He admitted that Dr. Fitchett had not given an untrue certificate, but found that he had violated the general concept of duty of care as it was laid down in English law.

The relationship between the plaintiff and defendant was that of doctor and patient. The doctor knew—he admitted that he knew—that the disclosure to his patient of his opinion as to her mental condition would be harmful to her. He was careful not to tell her directly what that opinion was. Nevertheless, he wrote out and gave to Mrs. Furniss's husband a certificate expressing that opinion. If he ought reasonably to have had in contemplation that Mrs. Furniss might be injured physically as the result of his giving that certificate (and that on the evidence is beyond dispute), then he was under a duty to take care to avoid an act which he could reasonably foresee would be likely to injure her. On the facts, if Mrs. Furniss was to be confronted by this certificate, it was likely to do her harm. The judge made the following statement:

In these circumstances, it seems to me not only likely, but extremely likely, that when the husband was

charged by his wife with mental instability he would be goaded into a "tu quoque" retort and that he would disclose to her either the certificate or at all events its contents. That he apparently did not disclose it and that the certificate remained hidden from Mrs. Furniss for a whole year speaks volumes for the husband's restraint. It is also to be noted that in giving the certificate to Mr. Furniss the doctor placed no restrictions on its use. It was not even marked "confidential". On that evidence I can only conclude that Dr. Fitchett ought reasonably to have foreseen that the contents of his certificate were likely to come to his patient's knowledge and he knew that if they did they would be likely to injure her in her health.

I find that the defendant doctor was aware that the opinion which he expressed in the certificate of 21st May 1956 would, if it should come to the knowledge of his patient, be likely to injure her in her health. I find also that in the circumstances in which he issued that certificate—handing it to the patient's husband to be given to his solicitor, knowing that husband and wife were then estranged, and without marking it as confidential or otherwise restricting its use—he ought reasonably to have foreseen that the certificate or its contents would be likely to come to the knowledge of his patient. I conclude, therefore, that in the circumstances to which I have referred the doctor owed to his patient at common law a duty to take reasonable care to ensure that no expression of his opinion as to her mental condition should come to her knowledge. The doctor did not take any precautions in that respect and in my opinion a cause of action was thus disclosed in the statement of claim and in the evidence adduced in support of it.

The motion for judgment for the defendant therefore fails; but before leaving that topic I would point out that the duty which I have held the doctor to owe to his patient in this case is far less extensive than the duty which is laid on a medical practitioner by the Hippocratic Oath and by the Code of Ethics of the British Medical Association. In some future case it may be necessary to determine whether, subject to some exceptions such as those adverted to in my summing up and others that readily suggest themselves, the duty to preserve a patient's secrets may not be much more extensive than the duty I have here held to exist and approximate very closely to the duty described in the British Medical Association's code of ethics. I venture to express the hope, and the belief, that such is indeed the law; but it is unnecessary in the present case for me to decide whether my belief is justified. Sitting, as I am, as a Judge of first instance I ought not unnecessarily to express any concluded opinion on that point.

It may now, perhaps, be time to attempt a summary of what has been said and produce some rules for the guidance of practitioners.

1. A doctor must take care not to give a third party a certificate as to the patient's condition if he can reasonably foresee that it might come to the patient's knowledge and cause him physical harm.

2. The rule is not absolute and depends on circumstances. Take the case of a doctor who discovers that his patient entertains delusions in respect of another, and in his disordered state of mind is liable at any moment to cause death or grievous bodily harm to that other. Perhaps the public interest requires him to report that finding to some one. Take the case of a patient of very tender years or of unsound mind. Common sense and reason demand that some report on such a patient should be made to the patient's parent or other person having control of him. But public interest requires that care should be exercised in deciding what shall be reported and to whom. Publication or communication of the report to other than appropriate persons could still be a breach of the duty owed by the doctor if the patient thereby suffers unnecessary physical harm. In certain circumstances the issue of a certificate might have resulted to the benefit of the patient. Indeed, that was Dr. Fitchett's own contention. He thought that as a result of it Mrs. Furniss would not be committed to a mental institution without prior examination by a specialist in psychiatry.

3. The doctor can cover himself to some extent in a jury's eyes by marking the certificate "confidential",

supposing always, of course, that it is given to an appropriate person and for good cause.

4. The doctor's duty not to disclose is much less strong when the certificate is given to a public authority, which has perhaps some shadow of right to the disclosure, than it is when given to a husband, wife or relative who might leave it about or show it. There must, however, be good cause for the disclosure, and it must be made in the interests of the patient rather than in the public interest.

5. A doctor may owe a duty of care not to disclose directly to the patient, if he foresees the latter may suffer physical injury in consequence. This is a very delicate principle considered in the abstract and divorced from concrete instances. A doctor will have to exercise discretion.

Finally, it should be emphasized that the doctor's primary responsibility is to the patient, and the law invades this principle only to the extent to which society requires the patient's interests to be subordinated to its own. This is obviously a dangerous principle in anything other than a liberal society, but it is perhaps a workable one for our purposes tonight. The general conclusion, therefore, is that a doctor should disclose as little as possible. *Fitchett v. Furness* is not a satisfactory guide as to principle, because here was the exceptional case in which the doctor had admittedly been indiscreet in writing the certificate. The case of the paranoid likely to murder his supposed persecutor is a much more borderline case, in which hitherto the authorities have remained silent.

#### FLUOXYMESTERONE ("HALOTESTIN") IN ADVANCED BREAST CARCINOMA.

By BASIL A. STOLL, F.F.R., M.R.C.S. (London),  
*Peter MacCallum Clinic, Melbourne.*

TESTOSTERONE was first reported in the treatment of breast cancer in 1939, two years before Huggins reported on the treatment of prostatic cancer by stilboestrol. Testosterone propionate has been the androgen extensively used for the past ten years. For this reason new androgens are best compared with it, both for clinical efficacy and for side effects. Adair and other authors reported the effect on bone metastases of testosterone propionate, at a dosage of 1200 milligrammes monthly. They noted relief of pain within two weeks in the majority, but recalcification in less than 20% of the cases (Table I). Relief of symptoms was occasionally prolonged for two or more years. In the treatment of metastatic skin nodules from breast carcinoma, the results are less satisfactory, as only 15% to 20% of cases show some temporary regression of nodules. Visceral metastases in other organs, e.g. lung and pleura, may

TABLE I.  
Reports in the Literature on the Objective Benefit from Testosterone.\*

Authors.	Number of Cases.	Metastases.		Pre-Menopausal.	Post-Menopausal.
		Skeletal.	Soft Tissue.		
Adair, 1949 ..	102	19	15	—	—
American Medical Association Council, 1949	159	18	20	—	—
Pearson <i>et alii</i> , 1955 ..	167	—	—	13	20
Peters, 1955 ..	282	—	—	11	17
Pyrh�, 1955 ..	67	—	—	17	19

\* Figures refer to percentage of cases in which benefit was observed.

regress on testosterone, but such metastases often progress while metastatic skin nodules or osseous metastases are regressing. The objective benefit from testosterone therapy appears, from Table I, to be somewhat greater in the older age group.



## Choice of Androgen.

Natural testosterone (bull androgen) was the first androgen to be used clinically. Unfortunately, testosterone taken by mouth is broken down by the liver into relatively

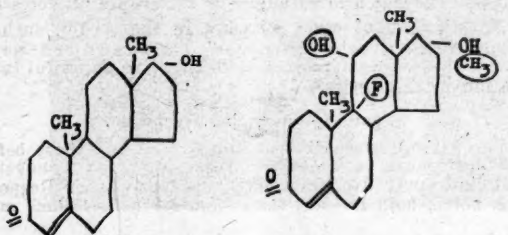
duration of action. A further disadvantage is that their action cannot be terminated if hypercalcaemia is induced. Nevertheless, these preparations and also the phenyl propionate and the enanthate are of value for maintenance therapy when an adequate blood level has been built up.

TABLE II.  
The Metabolism of Androgens: Urinary Excretory Products.

Androgens.	17-Ketosteroid Excretion.	Urinary Creatinine.
Testosterone propionate ..	+	±
Testosterone sublinings ..	±	±
Methyl testosterone ..	±	+
"Halotestin" ..	±	+

<sup>1</sup> Five times as potent as methyl testosterone in androgenic and anabolic activity. Retention of nitrogen, phosphorus, potassium and calcium, but little sodium retention.

ineffective compounds such as androsterone. When injected in oily solution, testosterone is rapidly eliminated from the body. In order to overcome this rapid elimination, various esters of testosterone have been used, and a solution of



Synthesized in 1935; used in treatment of mammary cancer in 1939 (Loesser).

9-Fluoro-11-hydroxy-17-methyl-testosterone

## TESTOSTERONE

## "HALOTESTIN"

FIGURE I.

Comparison of the structure of testosterone and "Halotestin".

testosterone propionate in oil has been, until recently, the common form of androgen administered for mammary carcinoma. Unfortunately, it is necessary to repeat injections

A punch card form for analyzing results. It contains various fields for patient information and treatment outcomes, including AGE, DURATION, ONSET, HISTORY, NAME, RESULTS, LOCAL IMPROVEMENT, THERAPY, and DISCONTINUATION. Each field is represented by a grid of circles, some of which are punched out.

FIGURE II.

Punch card for analysis of results.

at intervals of 48 to 72 hours in order to maintain an adequate blood level of androgen.

Depots of the crystalline suspension and also implants have therefore been tried, but have an indeterminate



FIGURE IV.

Response of an ulcerative chest wall to "Halotestin".

Because it is more stable, the less potent androgen methyl testosterone was introduced by Foss in 1939, and has been used orally instead of testosterone. When

TABLE III.  
The Effect of "Halotestin" on Calcium Balance.

Therapy.	Number of Patients.	Excretion Level.	Clinical Result.
"Halotestin" (short period)	5	Fall in five.	Relief in one.
"Halotestin" (long period)	5	Fall in four.	Relief in four.
On withdrawal of "Halotestin"	2	Remains high.	Relief maintained.
Stilbestrol substituted for "Halotestin"	1	Fall.	Relieved.
Prednisolone substituted for "Halotestin"	1	Fall.	Relief maintained.

swallowed it has about one-quarter the efficacy of testosterone propionate injected intramuscularly, but when absorbed sublingually the hormone is more efficacious. Unfortunately, it has the disadvantage of occasionally inducing jaundice.

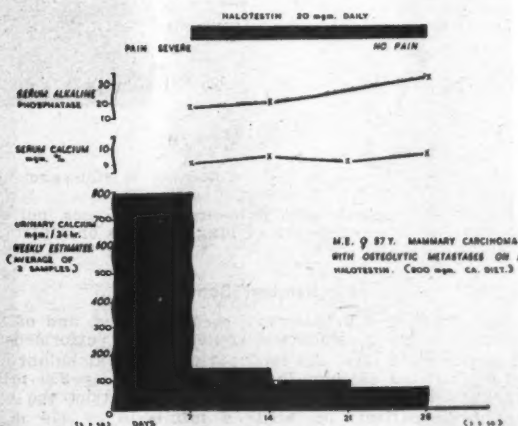


FIGURE V.

Calcium and enzyme control of response to "Halotestin".

The metabolism of methyl testosterone differs from that of testosterone propionate (Table II). The former increases creatinuria and is not excreted as 17-ketosteroids. "Halotestin" or fluoxymesterone, a new fluorinated methyl testosterone taken orally, shares these biochemical properties of methyl testosterone. "Sublings Testosterone", an orally absorbed form of testosterone, on the other hand is metabolized like testosterone propionate. Figure I shows the formulae of the two compounds compared.

#### Analysis of Cases.

A series of 50 patients have been treated with "Halotestin". The results were analysed by a punch card based upon a tabular survey (Figure II). There were 22 cases of bone metastases in patients whose response to treatment could be analysed. Subjective response (that is relief of pain) was noted in 18 cases (82%) and positive recalcification of metastases (without simultaneous X-ray therapy) in three cases. In one case recalcification of metastases appeared as early as six weeks, but in the others about eight to ten weeks after therapy was commenced (Figure III). The duration of improvement was nine months or longer in three cases, but in the majority it was three to six months only. These results are similar to those of testosterone propionate.

There were 29 patients with soft tissue growth involving breast, nodes or cutaneous nodules. Objective response was noted in eight cases (27%), possibly a somewhat better response than would be expected from testosterone propionate. The primary growth shows the most ready response, even of recurrent tumour (Figure IV), and in some patients who showed response in the primary growth, involved nodes did not respond at the same time. The duration of improvement was six months or longer in three cases, and improvement is still proceeding in these, although "Halotestin" has been withdrawn.

urinary calcium excretion for four days before and four days after "Halotestin" was instituted. A fall was noted in the five cases, but there was concomitant clinical benefit in only one case. In a further five cases, observations were continued for at least four weeks after "Halotestin" was instituted. In four cases there was a substantial fall in calcium excretion, and in all these there was clinical benefit (Figure V is typical). The fifth case showed neither a biochemical nor a clinical response. In four cases, urinary and serum calcium investigations were performed when "Halotestin" was discontinued. The four showed raised calcium excretion levels, although three patients were free from pain. Two of these showed no change in the calcium excretion as a result of the withdrawal of "Halotestin". In the third case, stilboestrol caused a rapid fall after one week (Figure VI), and in the last case prednisolone caused a fall after three weeks (Figure VII).

It was concluded that urinary calcium estimations do not parallel clinical remissions in these cases. A fall in urinary calcium excretion may occur (presumably due to the calcium-retaining properties of "Halotestin"), with no concomitant clinical improvement. On the other hand, there were two patients with marked relief of symptoms who nevertheless had persisting high urinary calcium levels. However, estimations may be useful for judging the stage at which to withdraw or reinstitute "Halotestin".

Investigation of other enzymes in the serum, such as copper-resistant acid phosphatase (Fishman) and serum phosphohexose isomerase (Bodansky) gave no useful information in this series.

#### Factors Influencing Response.

The patients were divided into two groups, those before the menopause or up to five years after the menopause, and those over five years after the menopause. Response was noted, both for soft tissue and for bone lesions more

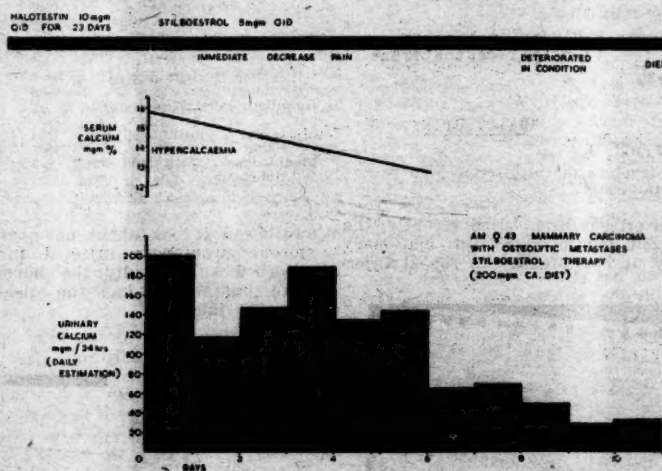


FIGURE VI.  
The effect of stilboestrol after "Halotestin" on biochemistry.

Out of five patients with pulmonary metastases, not one showed objective regression of lung opacities or control of pleural effusion.

#### Biochemical Control.

Investigation of urinary and serum calcium, and of the serum alkaline phosphatase content, was performed in 14 cases (Table III). All later patients were ambulant and on a restricted calcium intake (approximately 800 milligrammes daily), and it was decided to restrict the estimation to patients excreting a minimum of 250 milligrammes of calcium daily. Estimations were made both on the institution of "Halotestin" and on its withdrawal. Only one patient developed hypercalcaemia on "Halotestin" therapy. In five cases the observations compared the

or less equally in both groups, but the figures are too small for significance.

The effect of increasing the dose was assessed. "Halotestin" is said to be five times as potent as methyl testosterone, and therefore 15 to 20 milligrammes daily should be an effective therapeutic dose. Less than half the patients were treated at this level, and the rest at a dose of 25 to 40 milligrammes daily. There seems to be a definite improvement in the results for soft tissue metastases by increasing the dose above 20 milligrammes daily (Table IV). In two elderly patients the dose had to be reduced to 10 milligrammes daily because of intolerance, with headaches and nausea.

The response was compared to that from previous hormone therapy. It would appear that a renewed response



ILLUSTRATIONS TO THE ARTICLE BY BASIL A. STOLL, F.F.R., M.R.C.S.

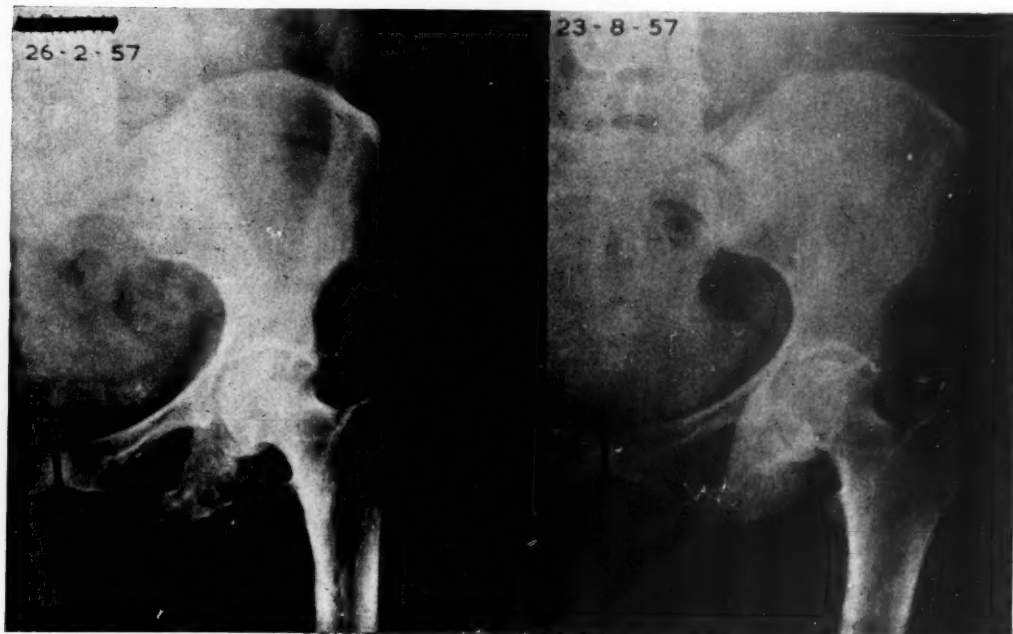


FIGURE III.  
Recalcification of the left ischium on "Halotestin".

ILLUSTRATIONS TO THE ARTICLE BY WILLIAM J. McCANN, F.R.C.S., F.R.A.C.S.

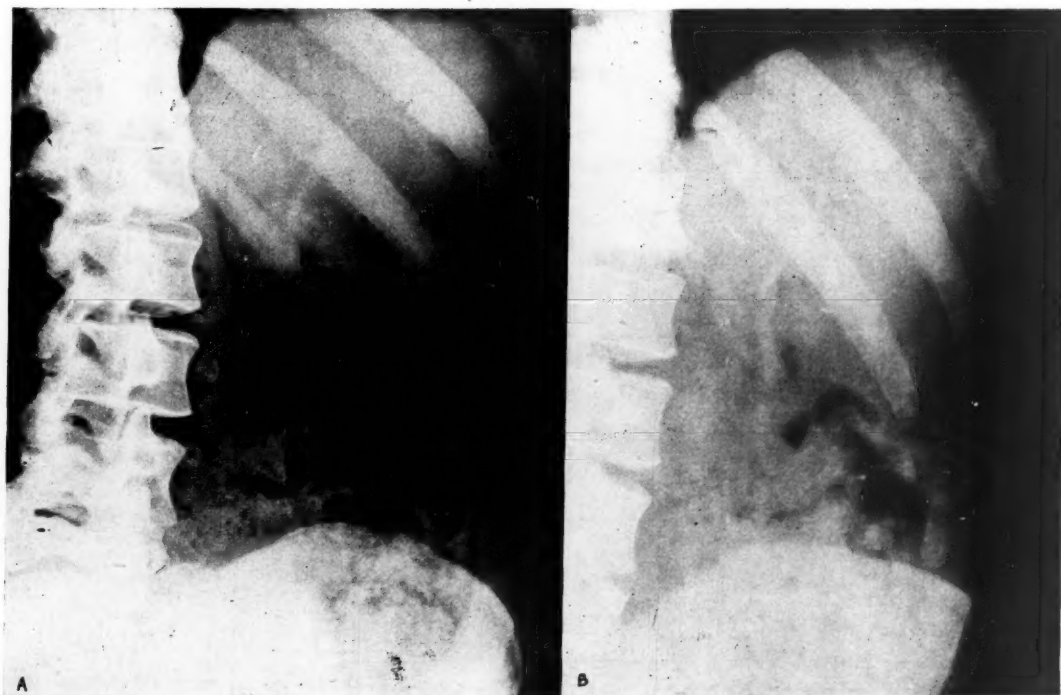


FIGURE IVA.

FIGURE IVB.

ILLUSTRATIONS TO THE ARTICLE BY A. T. H. JOLLY.



FIGURES IVA AND IVB.—Antero-posterior and lateral skiagrams of the foot taken on July 20, 1957, after the circulation had been restored for nearly five months. The calcified dorsalis pedis and diseased fourth metatarsal are clearly visible. FIGURES VA AND VB.—Antero-posterior and lateral skiagrams of the foot taken on November 28, showing resorption of diseased bone. The dorsalis pedis artery is less obvious than in the skiagrams taken on July 20.

ILLUSTRATIONS TO THE ARTICLE BY N. J. GRAY AND L. I. TAFT.

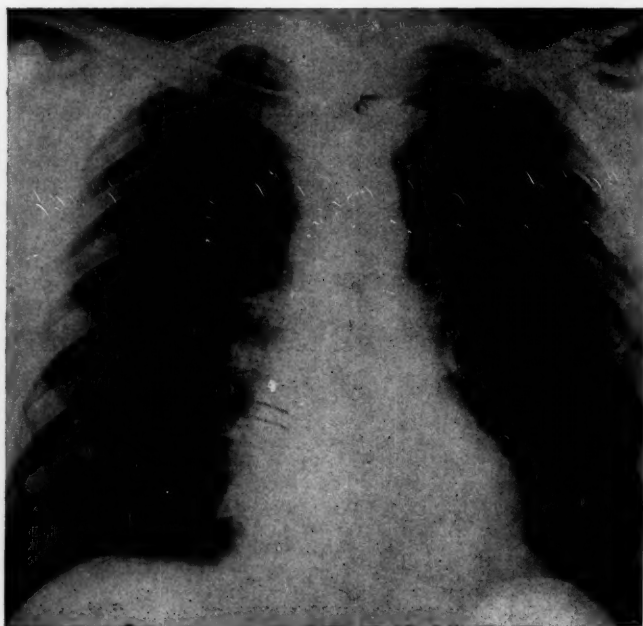


FIGURE I.

Skiagram of the chest taken 12 days after the patient's admission to hospital, showing miliary seeding throughout the lung fields.



FIGURE II.

Liver biopsy taken 24 hours after the patient's admission to hospital, showing two granulomata consisting of aggregates of lymphocytes and epithelioid cells, and with central necrosis. A giant cell can be seen at the left hand edge of the central granuloma; these granulomata contained acid-fast bacilli. (Hæmatoxylin and eosin stain,  $\times 200$ .)



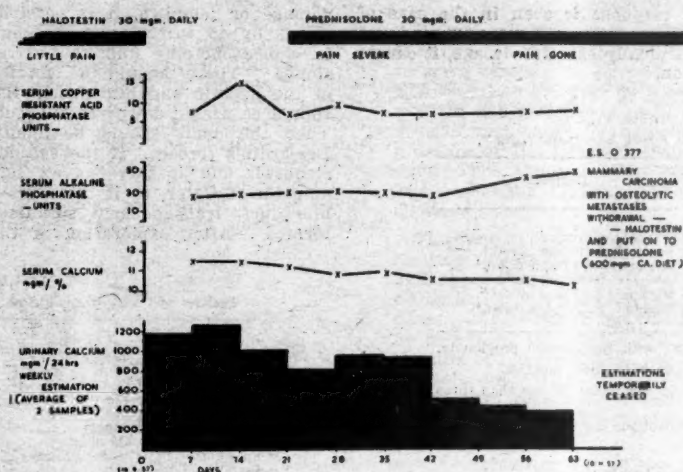


FIGURE VII.

The effect of prednisolone after "Halotestin" on biochemistry.

may be expected from patients previously responding to other types of androgen or corticosteroids, in whom the response has been lost (Table V). A response may also be expected particularly in the case of bone metastases, even when previous X-ray induced menopause or oestrogen therapy has failed.

#### Side Effects.

Side effects are minimal in general. The absence of masculinization, even with good clinical response, is remarkable. Figure VIII is a photograph of one patient

dose to this level. Seborrhoea and a receding temporal hair line were noted in one patient after six months on low dosage.

Weight changes were not constant. Twelve patients lost an average of four pounds in weight during the treatment, and three gained an average of nine pounds in weight. A further three patients showed a preliminary rise of three

TABLE IV.  
The Effect of Increased Dosage of "Halotestin".

Dose.	Numbers Showing Response in	
	(a) Bone Metastases.	(b) Soft Tissue Metastases.
15 to 20 milligrammes daily .. ..	7/10	1/10
25 to 40 milligrammes daily .. ..	11/12	7/19

whose breast carcinoma regression was shown graphically in Figure V. There is no obvious hirsuties in this or similar cases. Minimal hirsuties (of the upper lip mainly) and slight hoarseness were noted in about 25% of patients

TABLE V.  
Response of Metastases in Relation to Previous Hormone Sensitivity.

Previous Treatment by Induced Menopause, Androgens, Oestrogens or Corticosteroids.	Numbers Showing Response to "Halotestin".		
	Bone Lesions.	Soft Tissue Lesions.	Total.
Beneficial .. ..	8/10	8/10	16/20
Not beneficial .. ..	7/11	2/19	9/30

(Table VI). The proportion appeared to be somewhat higher when doses of over 25 milligrammes daily were given or when dosage was prolonged. In addition, nausea was noted in two cases, irritability, diarrhoea, oedema of the ankles, headaches and increased libido in one case each. In all cases these were noted at dosages higher than 15 milligrammes daily, and were relieved by lowering the



FIGURE VIII.

Typical absence of hirsuties on "Halotestin" (see Figure V).

to four pounds in the first month, followed by a fall. The metabolic effect is therefore not constant. Erythropoiesis is stimulated as with all androgens, and the haemoglobin level tends to rise.

#### Summary.

"Halotestin" gives clinical benefit similar to testosterone propionate in the case of bone metastases from mammary carcinoma. The results of calcium balance studies cannot always be correlated with the response of bone metastases.

Occasional spectacular response is seen in the case of soft-tissue metastases. The most obvious advantage of "Halotestin" is that, at clinically effective dosage, it causes little or no masculinization.

TABLE VI.  
Incidence of Side Effects from "Halotestin".

Side Effect.	Degree.
Hirsuties .. ..	Marked, one case. <sup>1</sup> Minimal, 3/19 (low dose). <sup>2</sup> Minimal, 7/20 (high dose). <sup>3</sup>
Hoarseness .. ..	Minimal, 3/19 (low dose). <sup>2</sup> Minimal, 2/20 (high dose). <sup>4</sup>

Previous treatment with testosterone propionate.

<sup>1</sup> All treated for more than three months.

<sup>2</sup> Includes three under treatment for less than three months.

<sup>3</sup> Both under treatment for less than three months.

#### Acknowledgements.

I wish to express my thanks to Dr. R. W. Talley, of the Upjohn Company, for free supplies of the drug under trial, and for willing help and advice throughout the trial. I wish also to thank the Organon Laboratories for a free supply of "Sublings Testosterone", which was used for a control series and found to have the same level of efficiency as intramuscular testosterone propionate.

#### THE FAILURES OF CHOLECYSTECTOMY.

By WILLIAM J. McCANN, F.R.C.S., F.R.A.C.S.,  
Prince Henry's Hospital, Melbourne.

To all with any experience of medical practice, it must be apparent that whilst the operation of cholecystectomy in the great majority of cases is performed with a low mortality (0.5%) and a gratifying result, there are a number of cases in which there is no improvement and indeed there may be an aggravation of the symptoms. The results of cholecystectomy will depend to a certain degree on the pathological features indicating operation. For non-calculous chronic cholecystitis, the results are disappointing. The mortality rate is low (0.5%), but the morbidity rate is high and less than 50% of the patients are symptom free. For calculous chronic cholecystitis (mortality rate 0.5%), a good result will be obtained in 90% to 95% of patients (Maignot, 1957). For acute cholecystitis, the mortality rate is probably about 3%, but this of course will be high if it is performed only in desperate cases.

I shall endeavour to deal with these failures. In particular, I wish to deal with the high morbidity rate after cholecystectomy for chronic cholecystitis without calculi. I do not intend to do more than mention that missed pathological conditions such as gastric neoplasm, peptic ulcer, hiatus hernia, cirrhosis, carcinoma of the pancreas, right renal disease, neoplasm of the colon and abdominal aortic aneurysm, etc., will not be relieved by cholecystectomy, and no surgeon should omit exploration of the whole abdomen at the time of operation.

The most frequent causes of pain occurring after cholecystectomy are: (i) stones in the bile duct; (ii) stricture; (iii) pseudo gall bladder; (iv) malfunction of the sphincter of Oddi; (v) pancreatitis (Table I).

#### Stones in the Bile Duct.

Of people who have been operated on for gall stones, autopsy showed stones in the duct in 16% (Young, 1947). So we see that residual stones in the common bile duct are the most likely cause of symptoms after cholecystectomy for calculous cholecystitis. If there is no liver

damage or jaundice, it is possible to show most of the stones by an intravenous cholangiogram. The treatment is choledochotomy and the extraction of the stone or stones. There should be forcible lavage with saline of the hepatic and bile ducts. Bakes graduated dilators should be passed down the bile duct to dilate the ampulla. Then the ducts should be finally searched again with Desjardin's forceps. If the sphincter of Oddi is tight or stenosed, and if there is an impacted calculus in the ampulla of Vater, or if there are recurrent stones in the bile duct, transduodenal sphincterotomy should be performed. After exploration of the bile duct, a "Latex"

TABLE I.  
Failures of Cholecystectomy Seen from 1952 to 1957.

Diagnosis.	Number of Cases.
Stones in the bile duct .. .. .	42
Strictures of the bile duct .. .. .	49 <sup>1</sup>
Spasm or stenosis of the sphincter of Oddi .. .. .	17
Pseudo gall bladder .. .. .	7
Recurrent pancreatitis .. .. .	5
Total .. .. .	120

<sup>1</sup> Thirty-five cases in London, 12 in U.S.A., two in Melbourne.

rubber T-tube is left in the common bile duct for 14 days—the tube being removed only if clamping it produces no discomfort or external biliary drainage, and if the post-operative cholangiogram shows free passage of dye into the second part of the duodenum. Again, if the bile drainage fluid is infected and not a good colour, culture should be performed and the duct irrigated with appropriate antibiotics. I shall not deal further with this obvious cause of post-cholecystectomy trouble, but shall now pass to the lesser recognized, but still not uncommon, sequelae of cholecystectomy.

#### Strictures.

Of strictures of the bile ducts 80% to 90% are man made (Catell, 1947; Walters, 1950)—the result of surgical trauma; 70% of these patients are less than 50 years of age, and approximately 25% are in the fourth decade of life (Gray, 1951). In the greater proportion of cases, the injury may be due to: (i) poor exposure; (ii) carelessness; (iii) lack of anatomical knowledge; (iv) hæmorrhage—cystic artery and right hepatic artery; (v) traction and application of the clamp to the angulated hepatic duct and bile duct, and failure to display properly the junction of the cystic duct and common hepatic duct and common bile duct; (vi) injury to the bile duct during exploration; (vii) leakage of bile and pooling, producing a sclerosing effect; (viii) damage to the blood supply of the common bile duct (Figure 1).

The injury to the bile duct may be noticed at the time, but usually the poor exposure, the pool of blood, etc., help to conceal the error temporarily. However, in the post-operative period it reveals itself as obstructive jaundice, external biliary fistula or biliary peritonitis. Less often when there is a partial constriction of the bile duct, some months and even years may elapse before it reveals itself with attacks of slight jaundice, pain, fever and loss of weight.

In the common type seen post-operatively, after careful preparation with protein, blood, vitamin K, electrolytes, etc., further operation takes one of the following forms. If possible, end-to-end union of the duct is effected over a T-tube. If the lower end is not suitable or cannot be found for this procedure, then an hepatico-jejunostomy or choledoch-jejunostomy may be performed, or else an hepatico-duodenostomy or choledoch-duodenostomy (Figure II).



If the stricture is partial, either a Heinecke-Mikulicz type of repair *plus* T-tube or dilatation *plus* T-tube may be the procedure. In these operations it is important to have mucosa-to-mucosa approximation to lessen the likelihood of stenosis developing later; this is the serious complication of the operation and is heralded by recurrent attacks of ascending cholangitis; it is stenosis with obstruction and not regurgitation that causes the trouble. For this reason numerous prosthetic tubes of bouncing clay (silicone), vitallium, polythene or rubber have all been left in the site of anastomosis; most are passed a few weeks after operation, but those that are not passed within

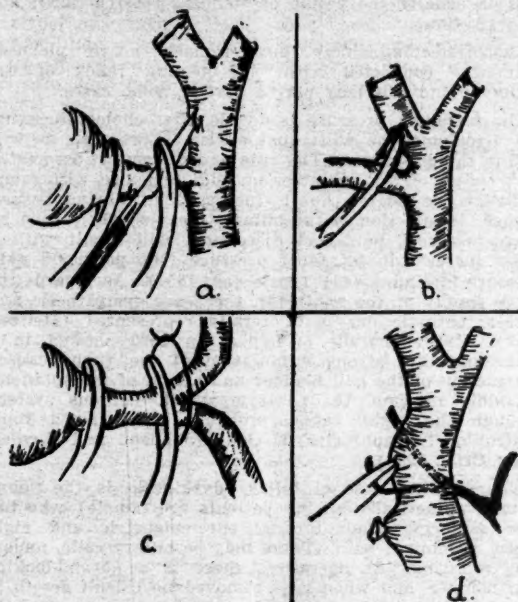


FIGURE I.

Injuries to the bile duct: (a) nicking the hepatic duct when cutting the cystic duct; (b) forceps on the cystic duct have nipped the right hepatic duct; (c) the junction of the common hepatic and common bile duct angulated under tension and caught in the forceps with the termination of the cystic duct; (d) "going for" the severed cystic artery in a pool of blood and nipping the hepatic duct, too.

months or years become thrombosed with bile debris and have to be removed by further operation. Technically, the suture of mucosa of the intestine to mucosa of the bile duct with interrupted sutures is probably the most important step to delay the onset of stenosis of the anastomosis. In large series, satisfactory results were obtained in about 80% of patients, but 10% to 20% of the patients (Walters, 1953) have to be reoperated on. The chances of a good result diminish and the operative risk increases with each additional operative procedure. The operative mortality rate is about 7%.

The incidence of bile-duct stricture in Victoria would appear low when compared with the 100 patients a year operated on at the Lahey Clinic in Boston, and with the large numbers at the Mayo Clinic, and other centres in the U.S.A. In England under the National Health Scheme, where surgery is performed only by trained surgeons, I am sure the relative incidence is much lower than in the U.S.A. In Australia the strictures are dispersed amongst a large number of surgeons, so that probably each one sees no more than three or four in his career. From my experience, this is the reason for the condition being an apparent, rather than a real, rarity here as compared with centres abroad.

### Pseudo Gall Bladder.

Pseudo gall bladder is a condition again man made, and is the result of a long stump of cystic duct being left and, in some cases, even part of the neck of the gall bladder. The condition presents with recurrence of attacks of cholecystitis, and sometimes small calculi may be present. The attacks may also be associated with jaundice due to pressure of the inflamed swollen remnant on the hepatic duct. Thus this condition may also produce a stricture. The remnant may show up in an intravenous cholangiogram.

The treatment is excision of the pseudo gall bladder and ligation of the cystic duct flush at the bile duct. In some difficult cholecystectomies, e.g., acute cholecystitis, it may be better to run the risk of pseudo gall bladder than to damage the bile duct, but in most cases the condition could be prevented by careful exposure of the three ducts and ligation of the cystic duct one quarter of a centimetre from the bile duct. This complication is more likely to follow when retrograde cholecystectomy (that is, beginning at the fundus of the gall bladder) is performed.

### Malfunction of the Sphincter of Oddi.

Stenosis, odditis and biliary dyskinesia are a few of the names which have been used for spasm of the sphincter

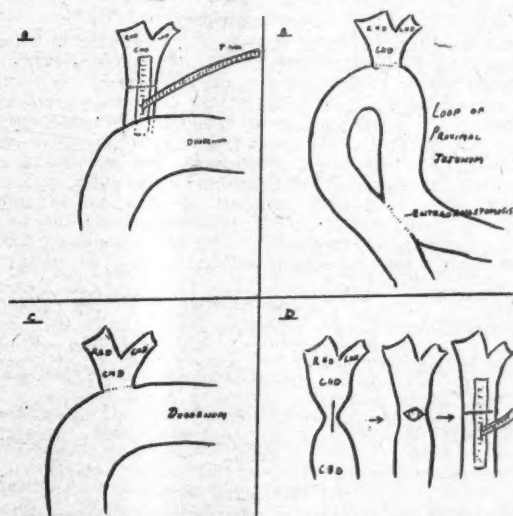


FIGURE II.

Operations for stricture of the bile duct. A, choledochostomy, and end-to-end repair over a T-tube; B, hepatico-jejunostomy; C, hepatico-duodenostomy; D, Heinecke-Mikulicz repair for partial stricture.

of Oddi. The term biliary dyskinesia literally means "a disturbance in the normal kinetics of bile flow", and I am sure most medical practitioners view it as a rather nebulous condition or as the diagnosis a destitute consultant may make. Indeed, it is only in the last three years that I myself have seen the light, and have endeavoured to understand, to diagnose and to treat confidently this condition. We must first consider the physiology of bile flow. The bile is secreted by the liver into canaliculi, and then goes to the right and left hepatic ducts, to the bile ducts, to the gall bladder and down to the ampulla of the bile duct, and to the sphincter of Oddi. This *vis a tergo* from the liver is at a pressure of 60 to 70 millimetres of water, and is modified by the resistance of the sphincter of the biliary tract, the cystic duct, the neck of the gall bladder, and possibly of an hepatic duct (Mirizzi, 1940).

The important regulating mechanism of the bile flow is the sphincter of Oddi. This is a complex structure which actually consists of a biliary, pancreatic and ampullary component (Figure III). The sphincter of Oddi normally is spastic in the fasting state, and withstands a pressure of 100 to 150 millimetres of water. The normal secretory pressure of the liver cannot overcome the resistance and the bile is directed to the gall bladder. The volume and pressure in the gall bladder are constantly reduced by fluid absorption. The concentrating power of the gall bladder acts as a pressure-regulating mechanism (Lester and Colp, 1952). The entry of chyme into the duodenum causes contraction of the gall bladder and simultaneous

gradually dilates and the sphincter may lose its tone and the symptoms subside. Patients with a narrow bile duct are much more sensitive to pressure changes than those with a dilated duct, and so quite frequently the cholangiogram will appear normal and the bile duct will be of normal size at laparotomy. Normally an intraduct pressure of up to 500 millimetres of water is tolerated without pain (Pribram, 1950). The function of the sphincter is to regulate the flow of digestive juices (biliary and pancreatic) into the duodenum periodically (Reynolds, 1955). Prevention of the regurgitation of duodenal contents into the bile and pancreatic ducts is not its primary function (it has been shown experimentally that ascending cholangitis is usually the result of stenosis (obstruction), not regurgitation).

Cholecystectomy may also remove nerve plexuses intimately connected with the opening reflex of the sphincter and this may play a part in some cases.

The intraduct pressure is double after cholecystectomy, and also morphine will cause a rise in pressure, as will acid in the duodenum. The pain and discomfort associated with spasm of the sphincter of Oddi and also with stone occlusion are entirely due to increased intraduct pressure. If there is infection in the biliary tract, usually caused by *Bacterium coli*, pain and discomfort will result with a lesser increase in intraduct pressure (the perfusion pain pressure is diminished) (Sarkisian, 1954). As regards the nerve supply of the sphincter, there are sympathetic and parasympathetic nerves bilaterally represented (Meltzer, 1917). Experimentally, Westphal in 1923 showed in a guinea-pig that strong stimulation of the vagus caused contraction of the gall bladder and spasm of the sphincter of Oddi. In man, the parasympathetic nervous system, through the right vagus, probably maintains a tonic control of the sphincter of Oddi (Johnson and Boyden, 1943; Crile, 1951).

The classical case of biliary dyskinesia is the young woman (practically all my patients are female) who has been suffering from intermittent epigastric and right upper abdominal pain. There may be biliary colic, nausea and vomiting. At operation, there is a normal-looking gall bladder, and when it is removed no calculi are to be found. Within a short time she may get recurrence of the pain, and often it is more severe than before operation. Chills, fever and jaundice are not common. When she is seen in a year's time, she may already have had a laparotomy and division of adhesions. There may be some emotional disturbance and the attacks of pain may be related to the menses. Practically always fried foods (especially chipped potatoes), alcohol, and tomato and orange juice will precipitate attacks. In most cases one-sixth of a grain of morphine will provoke spasm and permit confirmation of the diagnosis.

The confirmatory diagnostic test (the morphine cholangiogram test) I have evolved is as follows. An intravenous injection of "Biligrain" (sodium organic iodine compound, sodium iodopamide) is given, and serial skiagrams are taken. When the bile duct has been demonstrated radiologically, usually about 30 minutes after this intravenous injection, an intramuscular injection of one-sixth of a grain of morphine is given. Further skiagrams are taken and also the patient's reaction is noted (Figure IV). In the classical cases of dyskinesia due to spasm, the morphine will induce an attack of pain within 30 minutes or so. This pain will be identical with the attacks complained of, and further X-ray pictures will show that there is now a dilatation of the bile duct.

My thesis is that morphine produces spasm of the sphincter as shown by the resulting dilatation of the bile duct, and that, in reproducing at the same time the identical pain, it shows that the symptoms are due to dyskinesia resulting from spasm of the sphincter of Oddi. If the test gives a positive result, then the diagnosis is definite. A negative result, due either to poor bile duct shadow or to failure to respond characteristically to morphine, does not rule out dyskinesia completely.

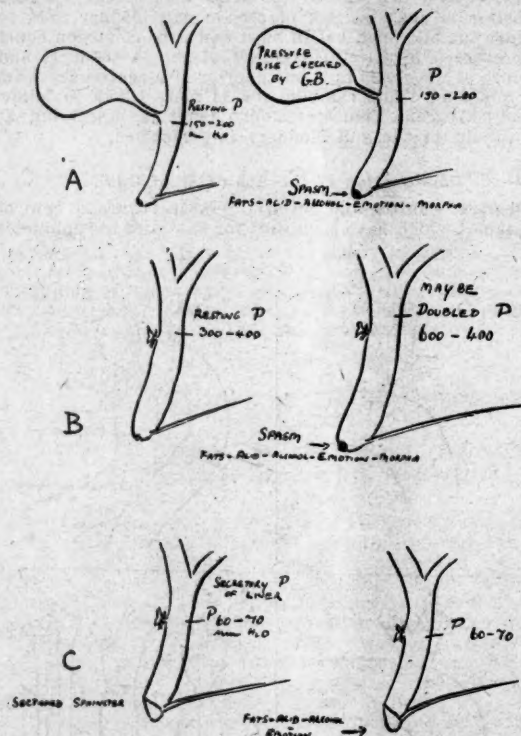


FIGURE III.

Spasm of the sphincter of Oddi: A, before cholecystectomy; B, after cholecystectomy; C, after sphincterotomy.

relaxation of the sphincter. If the sphincter mechanism fails to relax with the entrance of gastric contents into the duodenum, the biliary tree becomes distended, with resultant pain or biliary colic. The etiology of the spasm may be functional or organic. The functional spasm is seen in young, highly emotional women (in my small series four were divorced). In this group the pain is frequently related to the menses.

Spasm may also result from biliary tract disease or papillitis, or there may be an organic stenosis. Hyperacidity may also produce spasm; e.g., tomato juice or orange juice and also alcohol stimulate the production of acid. Morphine may precipitate an episode of pain by producing spasm in some people.

Dyskinesia will, of course, be greatly aggravated by cholecystectomy in these cases, as the pressure-regulating mechanism has been removed. This is borne out in the poor results of cholecystectomy for non-calculous cholecystitis; the loss of the pressure regulator and the spasm of the sphincter give an increase in intraduct pressure resulting in discomfort and pain. In some cases the duct



In the treatment of the condition, the target is the sphincter of Oddi. Medical treatment consists of correct diet (the avoidance of fats, lead drinks, foods which increase acidity, alcohol, coffee, spices). Antacids help by neutralizing the acid. Antispasmodics such as belladonna, atropine, "Pro-Banthine", "Felicur", "Merbentyl", etc., have not been proved helpful in my experience. Amyl nitrite, however, will give relief. Pethidine will relieve the spasm and pain, and in two cases addiction to this drug was imminent.

Many procedures have been proposed and performed for the relief of sphincter spasm. (i) Gradual dilatation with dilators gives a transient effect. (ii) The insertion of a T-tube into the duodenum through the sphincter gives a temporary effect. (iii) Choledcho-duodenostomy (Hosford, Bart's) and choledcho-jejunostomy frequently develop stenosis of the stoma and also do not relieve the pancreatic duct from the effect of spasm. (iv) Transduodenal sphincterotomy is the best procedure, as the endocholedochal operation is uncertain, and ampullary neoplasms may be missed (Doubilet and Mulholland, 1956). Sphincterotomy is the nearest approach both anatomically and physiologically to the normal state. In patients with a positive result to the morphine cholangiogram test, this operation has been uniformly successful. Crile (1951) of Cleveland had tried vagotomy, but with disappointing results and the unpleasant side effects of gastric retention, etc. (Mallet-Guy, 1953); he has now abandoned this operation for dyskinesia. In the case in

TABLE II.  
Results of Transduodenal Sphincterotomy.<sup>1</sup>

Indication.	Number of Patients.	Results.
Spasm, stenosis and biliary dyskinesia.	12	Nine good. One no better. One slightly improved. One operation too recent to assess. No death.
Recurrent or relapsing pancreatitis.	5	Three good (one pancreatic sphincterotomy). One no better. One death (acute pancreatitis).

<sup>1</sup> Sphincterotomy was successful in all the patients with a positive result to the morphine cholangiogram test. Five additional patients have been subjected to sphincterotomy without mortality since the paper was submitted for publication.

which the dyskinesia consists of stricturing of the terminal inch or so of the bile duct, sphincterotomy will not relieve the obstruction, and in this case, in which the supraduodenal part of the bile duct is dilated, a choledcho-duodenostomy would be preferable (Walters, 1956).

#### Pancreatitis.

As the last type of post-cholecystectomy disorder we have recurrent pancreatitis, in which the pancreatitis is the result in some cases of forcible regurgitation into the pancreatic duct, and in others of the obstruction of the opening of the main pancreatic duct—the result of spasm of the sphincter of Oddi or spasm or stenosis of the pancreatic sphincter. In these cases the head of the pancreas is indurated, and often the pancreatic duct is dilated and can be palpated along the pancreas. Spasm of the sphincter in many cases has been the initiating factor, and again sphincterotomy may be of value. The pancreatic duct can usually be seen opening into the medial aspect of the ampulla. It should be gently probed if the pancreatic duct is dilated, and in one case the opening was slit-relieving stenosis-pancreatic sphincterotomy with resulting cure of pain (Starr, 1953). The results in these cases are not as satisfactory, as the patient has usually been operated on a few times previously, is usually in an older age group, and probably has irreversible pancreatic pathological features. The patient is often a pethidine addict, and in every case should continue on a fat-free

diet and abstain from alcohol for at least a year. Choledcho-duodenostomy or choledcho-jejunostomy may be best again if the pancreatic part of the bile duct is strictured by the pancreatitis. Splanchnicectomy, insertion of the tail end of the pancreatic duct into the jejunum (retrograde drainage) and even partial pancreatectomy have been tried for intractable pancreatitis (Catell and Warren, 1953).

#### Conclusion.

Choledocholithiasis (stones in the bile duct) is the commonest cause of post-cholecystectomy symptoms. In this series, stricture of the bile duct is prominent, but this was due to the selective nature of the cases referred to Mr. Rodney Maingot. In Australia, bile-duct injuries are probably not as rare as one would think. The apparent rarity is probably due to the cases being dispersed amongst a large number of surgeons, unlike those in the centres abroad.

#### Summary.

The less recognized conditions of pseudo gall bladder and relapsing pancreatitis are mentioned. The understanding, diagnosis and treatment of the conditions resulting from malfunction of the sphincter of Oddi are facilitated. A definitive method of confirming the diagnosis of the entity of spasm of the sphincter of Oddi, or biliary dyskinesia, is presented and details are given of the morphine cholangiogram test.

#### Acknowledgements.

I wish to acknowledge gratefully the patient tuition and inspiration I received in the surgery of the pancreatic and biliary tract from Mr. Rodney Maingot during the years I was his assistant at the Royal Free Hospital, London. Also the Royal Free Hospital for sending me to the United States on a travelling fellowship and enabling me to glean knowledge first hand from Dr. Richard Catell of the Lahey Clinic, Boston, and from Professor Henry Doubilet of the Bellevue Hospital, New York. Also the staff of the Radiological Department of Prince Henry's Hospital, Melbourne, under Dr. Norman Long, for their cooperation in performing the morphine cholangiogram tests.

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#### Legend to Illustrations.

FIGURE IV.—The morphine cholangiogram test to confirm the diagnosis of spasm of the sphincter of Oddi or biliary dyskinesia. (A) Skiagram taken 30 minutes after the intravenous injection of "Biligradin" shows a normal-looking bile duct (posterior oblique view). (B) An intravenous injection of one-sixth of a grain of morphine was given to the patient after 40 minutes. Skiagram taken after 60 minutes shows the relative dilatation (distension) of the bile duct. At the same time the patient experienced an attack of biliary colic and deep back pain in the region of the eleventh or twelfth thoracic vertebra. This is a positive reaction to the test and the patient has had no symptoms since a transduodenal sphincterotomy was performed 18 months prior to the time of writing (March, 1955).

### Reports of Cases.

#### OBSERVATIONS ON DIABETIC GANGRENE AFTER RESTORATION OF CIRCULATION.

By A. T. H. JOLLY,

Midland Junction, Western Australia.

THE patient, a diabetic man, aged 69 years, was first seen on March 1, 1957. He had been in hospital for three months, finally leaving at his own risk rather than face his second major amputation. He was first diagnosed as a diabetic in 1938 and lost his left leg in 1943 with a mid-thigh amputation. At present he is receiving 28 units of protamine zinc insulin and 16 units of soluble insulin daily.

Examination revealed him to be an elderly obese male with a left mid-thigh amputation. The gangrenous fifth right toe had been amputated also, but the wound had never healed and was gaping with unhealthy bloodless granulations (Figure I). The fifth metatarsal had been removed at the mid-shaft, and its proximal half was lying in the floor of the ulcer, whilst a small part of the fourth metatarsal was exposed just below the metatarso-phalangeal joint. No dorsalis pedis, anterior tibial or posterior tibial pulse could be palpated in his foot or ankle. His popliteal pulse was weak, but his tactile and pain sensations were intact. His blood pressure was 160/90 millimetres of mercury. He was warned that anticoagulant therapy might not save his foot and that a mid-thigh amputation might be necessary. He elected to try anticoagulant therapy.

His prothrombin level was reduced to 20% of normal with 150 milligrammes of "Dindevan" daily. He was then given heparin, 5000 units twice daily, which further prolonged his accelerated clotting time and gave him a false prothrombin level of between 5% and 10% lower than his actual prothrombin level. This method is useful when attempting to restore circulation. If hæmorrhage occurs it can be controlled quickly by a few milligrammes of protamine sulphate given intravenously, whereas hæmorrhage from phenylindanedione and dicoumarol takes much longer in responding to vitamin K administration and can be serious.

Four days after combined treatment both the dorsalis pedis pulse and the anterior tibial pulse became palpable, and there was obviously great improvement in the circulation of his foot. The granulations bled freely if probed, and his foot was warm and pink. The necrotic fifth metatarsal was then removed under local anaesthesia and the skin flaps were pulled loosely together (Figure II). Healing occurred after two months, except for two sinuses

at either end of the scar (Figure III). These would close for a day or so and then discharge serous pus. The patient is a pensioner, and apart from four weeks in a small private hospital, all treatment was carried out at his home with the aid of a visiting Silver Chain sister.

As the patient had been confined to bed for seven months, it was decided to get him on his feet again for further investigation, and he attended the surgery for an X-ray



FIGURE I.  
The gangrenous foot prior to anti-coagulant treatment.

examination of his foot on July 20 (Figure IV). The antero-posterior view showed osteomyelitis of the fourth metatarsal and adjacent proximal phalanx, whilst the metatarso-phalangeal joint had been completely destroyed, with resorption of the metatarsal head and phalangeal



FIGURE II.  
The foot after restoration and removal of the fifth metatarsal. Note the sinus at the distal end of the scar.

base. Apparently the joint had become infected at some stage and chronic osteomyelitis had involved the bone, thus accounting for the intermittently discharging sinuses. Both antero-posterior and lateral X-ray views showed a calcified dorsalis pedis artery. Further X-ray pictures taken on November 28 (Figure V) showed more resorption of necrotic bone with healthy new bone formation, and the calcified dorsalis pedis artery was less obvious. This

offers a good opportunity for research; the X-ray examinations can be repeated every few months and the effects on the diseased bone and calcified artery noted. Other treatment that the patient received consisted of a diet free from animal fats, the complete banning of smoking, Buerger's exercises, antibiotics and dressings to his foot.

Since then the circulation has remained quite good, and the patient is now maintained on 150 milligrammes of "Dindevan" daily, plus 2500 units of heparin every second day, his prothrombin level being 20% of normal. If heparin was reduced below this minimum the dorsalis pedis artery ceased pulsating.



FIGURE III.

The foot completely healed. Owing to osteomyelitis of the fourth metatarsal the wound breaks down intermittently.

At present the patient can walk with the aid of a stick or crutch and wears an ordinary shoe on his once gangrenous foot. No further operative procedures are contemplated, as his movements are obviously very restricted and his foot fulfils all present requirements. At the time of writing the wound had been healed for three weeks.

#### Discussion.

It could have been argued that as the anterior and posterior tibial arteries were completely occluded with atherosclerosis and organized clot, anticoagulants could be of no possible avail. Yet in fact the anterior tibial and dorsalis pedis pulses returned four days after intensive anticoagulant treatment. As the vasodilatory effect of heparin would be unlikely to affect a calcified artery, it can only be concluded that the pulse returned because the occluded artery became patent over a period of four days, and that anticoagulants in some way reversed the thrombosis or atherosclerosis. This lends support to Duguid's theory that atherosclerosis is actually due to the laying down of small intimal thrombi.

#### Summary.

A case of diabetic gangrene with reestablishment of circulation by intensive anticoagulant therapy is described.

Observations of the effects of the renewed blood supply on calcified vessels and diseased bone are made.

The importance of small doses of heparin to maintain a good circulation is noted.

### ASPIRATION LIVER BIOPSY IN THE DIAGNOSIS OF MILIARY TUBERCULOSIS: REPORT OF A CASE.

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In some systemic diseases such as miliary tuberculosis, brucellosis and sarcoidosis, clinical examination, liver function tests and other investigations may give negative or non-specific findings, whereas liver biopsy is frequently

diagnostic (Scadding and Sherlock, 1948; King, 1952; Finckh et alii, 1953; Joske and Finckh, 1955). Sometimes the problem of pyrexia of unknown origin may be solved rapidly by liver biopsy, and the history of such a case is presented here.

#### Clinical Record.

The patient, an electrical engineer aged 45 years, was employed at a dried milk factory. He had neither a family history of nor a history of contact with tuberculosis. When seen initially in April, 1957, he gave a history of three weeks' persistent fever, the temperature having varied from 100°F. to 104°F. He also complained of central abdominal pain, mild headaches, cramps in the legs and transient diarrhoea. A course of penicillin had been given without effect. He appeared very ill, but clinical examination gave otherwise negative findings, as were the results of full blood examination, microscopic examination of urine, culture of the stools, blood agglutination tests for brucellosis and typhoid fever, and X-ray examination of the lungs. However, blood culture grew *Staphylococcus aureus*, which was later considered to be a contaminant. He was treated over the next 10 days with tetracycline and erythromycin given intravenously, followed by oxytetracycline and chloramphenicol given orally, and was later given streptomycin four grammes daily for only two days. This treatment produced no clinical improvement, and he was transferred to the Royal Melbourne Hospital on the thirty-first day of illness.

He was severely ill and drowsy, with a temperature of 105°F. He had severe ulcerative glossitis and perianal and sacral desquamation. The lungs were clinically clear, the liver was impalpable and fundi and testicles were normal. Laboratory findings were as follows: haemoglobin value was 12.4 grammes per 100 millilitres; white cell count, 8000 per cubic millimetre; cerebro-spinal fluid normal. The chest X-ray film showed very fine nodulation throughout both lung fields suggestive of miliary tuberculosis (Figure I); microscopic examination of sputum revealed a heavy growth of monilia. Cultures of sputum, gastric washings, urine, stools, cerebro-spinal fluid and blood and guinea-pig inoculations of sputum and blood gave negative results for *M. tuberculosis*.

Twenty-four hours after his admission to hospital liver biopsy was performed to establish a conclusive diagnosis. This revealed typical tuberculous granulomata with giant cells and central caseation (Figure II); 14 acid-fast bacilli were identified by staining with Ziehl-Neelsen's carbo-fuchsin. Acid-fast bacilli were also readily detected in serial sections stained with auramine rhodamine and viewed through the fluorescence microscope. A portion of the liver biopsy cultivated on Löwenstein's medium grew *M. tuberculosis* after 14 weeks.

A confident diagnosis of disseminated tuberculosis was thus made within 24 hours of the patient's admission to hospital, and treatment was begun immediately with streptomycin, isoniazid and cortisone. A dramatic response was observed; he felt better, his appetite and strength returned and the temperature fell to normal (Figure III). He thereafter made steady progress and gained five pounds in weight in six weeks. The Mantoux test gave a negative result at weekly intervals until the eighth week, when a weakly positive reaction was obtained with 1:100 old tuberculin.

After two months, the temperature was normal, and the chest X-ray film was clear. Liver biopsy was repeated and granulomata were again seen; these did not contain bacilli, there was little fibrosis, and necrosis was much less pronounced. A further liver biopsy five months later showed healing granulomata. No bacilli were detected and the culture gave negative results. Six months after the onset of his illness, he resumed light duties on maintenance chemotherapy and was in excellent health when last seen four months later.

#### Discussion.

It is a striking feature of this case that a conclusive diagnosis was provided by liver biopsy within 24 hours of the patient's admission to hospital. Whereas miliary

<sup>1</sup> Drug Houses of Australia. Fellow to the Clinical Research Unit.

<sup>2</sup> Working with the aid of a grant from the National Health and Medical Research Council of Australia.



tuberculosis was suggested by the histological picture, absolute confirmation was obtained from the presence of acid-fast bacilli in the sections and their ultimate culture from the biopsy fragment. In this case acid-fast bacilli were sufficiently numerous in the granulomata to be found in sections stained with Ziehl-Neelsen's carbofuchsin. Serial sections of the liver biopsy stained by auramine rhodamine (Gray, 1953) and viewed through the fluorescence microscope (Matthaei, 1950) also revealed acid-fast bacilli; this in experienced hands may prove to be a more sensitive technique. The practice of making cultures of portions of liver removed by needle biopsy has been reported by Hurley (1952) and by others (Rumball and Baum, 1952). On the present occasion it was of particular value as all the other cultures were negative, and it may be undertaken with advantage whenever doubt exists, even if it necessitates repeating the biopsy after viewing the histological sections.

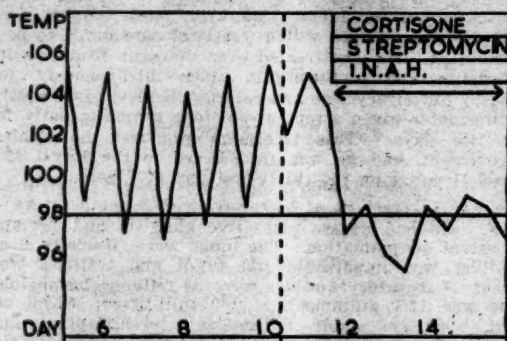


FIGURE III.

Temperature chart from the fifth to the tenth day of illness and then after the patient's admission to hospital. There was rapid response to treatment with isoniazid, streptomycin and cortisone.

The liver is usually involved in miliary tuberculosis, and biopsy may be performed with some confidence that granulomata will be found. Tubercles have been demonstrated in the liver by biopsy in patients with tuberculosis involving numerous other organs, particularly lungs, kidneys, adrenals, bones and joints (Haax and Van Beek, 1955; Olderhausen *et alii*, 1955).

The initial negative tuberculin response is a relatively common finding. The importance of this test naturally depends upon the age of the patient, and there are wide differences in tuberculin reactivity between various series presented in the literature, particularly in subjects with miliary or meningeal tuberculosis. Illingworth (1956) found that 42% of children in a group of 184 with miliary or meningeal tuberculosis gave a negative response with the tuberculin jelly test. A further analysis of this group showed that the results of the test were negative in 30% of those patients with radiological evidence of miliary infection in the lungs.

Miliary tuberculous infection is usually apparent in the lung films at a moderately early stage in the illness. However, as in this case, there may be a period of one or two weeks when the patient is seriously ill and the X-ray findings are inconclusive (Terry and Gunnar, 1957). It is during this phase that liver biopsy may reveal the cause of the illness, thus allowing full treatment to be instituted.

The use of cortisone in this patient was based on two premises. Firstly, while the results of cortisone treatment in chronic tuberculosis have been variable (Ford, 1957), some opinion favours its early use in cases of acute meningeal and miliary infection (Cocchi, 1956) in order to inhibit formation of avascular necrotic and fibrotic foci into which antituberculous drugs are slow to penetrate. Secondly, this patient had a temperature of 105°F., severe ulcerative glossitis and perianal desquamation. These are recognized complications of antibiotic therapy (Welch, 1954; Tolhurst *et alii*, 1955), and were regarded as hyper-

sensitivity phenomena which should benefit from steroid therapy. It is possible that the sudden fall in temperature and clinical improvement were mainly the result of cortisone administration, for treatment with antituberculous drugs alone usually produces a more gradual response.

The experience of this case indicates that, while the obvious diagnostic approach is a routine investigation by Mantoux test, X-ray examination of the lungs and sputum examination, some patients will require liver biopsy. The rapid and continued response to treatment, which included cortisone, was most gratifying.

#### Summary.

A rapid definitive diagnosis of miliary tuberculosis was obtained by needle biopsy of the liver in a patient with pyrexia of unknown origin.

A dramatic therapeutic response was obtained with combined antituberculous drugs and cortisone.

#### Acknowledgements.

We wish to express our thanks to our colleagues in the Clinical Research Unit for their assistance in the preparation of this paper. Miss E. Earle processed the histological material, and Mr. L. J. Swaby of the Public Health Department of the University of Melbourne reported on the auramine rhodamine stained sections. The photographs were prepared by Mr. E. Matthaei and the Visual Aids Department of the University of Melbourne.

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## Reviews.

*The Lost Days of My Life.* By Jane Simpson; 1958. London: George Allen and Unwin, Limited. 8½" x 5", pp. 169. Price: 15s. (English).

This is an autobiographical account of the experiences of a young girl admitted to a mental hospital at the age of 13 years, later certified and, after transfer to several similar institutions, discharged finally after ten years' continuous confinement.



While truth, of course, exists mainly in the eye of the beholder, the reader, as this sad chronicle unfolds, becomes uncomfortably aware that most, if not all, that is described herein can have been obtained only at first hand; this despite whether the picture is overdrawn or not.

It is stated that Jane was admitted, in the first instance, without justification; largely, it appears, as the result of the machinations of a malevolent mother who herself clearly suffered from a personality disorder of near psychotic proportions. There can be no doubt, however, that Jane herself was grossly disturbed, and that her admission, though possibly misguided, was justified as an attempt to rectify this. Be that as it may, what subsequently transpired provides a grim illustration, not only of man's inhumanity to man, but of the fact that bad treatment and lack of psychological understanding often make the patient worse.

*Post hoc* it appears that Jane was misdiagnosed as schizophrenic, and that this label, once firmly applied, determined in large part the course of subsequent events.

Even if the complete authenticity of this story is held in doubt, the psychological interplay between the various characters portrayed is of considerable interest, throwing as it does Jane's necessarily ambivalent attitude towards her mother into sharp relief. Thus Jane's feelings towards the sadistic "Nurse Evans" (later melodramatically strangled by another patient) contrast sharply with that expressed towards the ever-present, kindly but always ineffectual nurse whose sympathies appear to have been with Jane herself. In this, its least sensational aspect, Jane's story is of quite some interest.

**The Art of Clinical Refraction.** By Theodore H. Whittington, M.D., M.R.C.P., D.O.M.S.; 1958. London: Oxford University Press. 8½" x 5½", pp. 300, with illustrations. Price: 54s. 6d.

THEODORE WHITTINGTON's text-book on refraction can be recommended to all post-graduate students studying for the diploma in ophthalmology. Based on lectures given over the years to diploma candidates in London, it contains enough optics to give the student an understanding of lens systems without going into great detail, and this is combined with masterly clinical application.

The emphasis is on the practical application of refraction, which is regarded as just another part of clinical medicine. Clinical cases are quoted to demonstrate and emphasize the subject under discussion.

Refraction and its application to the patient cannot be learnt from a text-book. The out-patient department must provide the practice and experience; however, this text-book will make the student's task much easier and more pleasant.

The book is well produced, singularly free from error, and reasonably priced.

**The Introvert: A Psychiatric Study of Social Adjustment for Student and Layman.** By Ainslie Meares, M.B., B.S., B.Agr.Sc., D.P.M.; 1958. Springfield, Illinois, U.S.A.: Charles C. Thomas, Publishers. 9" x 5½", pp. 160. Price: 34s.

THIS book is primarily designed to help laymen understand difficulties in interpersonal relationships arising through introversion. In his preface the author explains that he does not discuss psychopathology. He believes that every introvert needs help in social adjustment, since he "is the stranger whom we know but whom we do not understand". The book aims to impart some knowledge to his relations, his friends and his teachers.

At the outset Dr. Meares lays himself open to a criticism of over-simplification. He refers to the 100,000 schizophrenics in America as introverts who have taken the wrong turning. It is implied that the type of handling recommended might have averted the catastrophe. Possibly such overstatements may be justified in an attempt to encourage relatives in their efforts, but in actuality there are 'introverts' and 'introverts'. There are different types of introversion with differing prognoses, whose unravelling presents a major problem to psychiatry.

Recognition of the introvert child commences in childhood.

From an early age the introvert child stands apart from other children on account of his shyness. While they are playing together he clings to his mother's skirt. He looks at them as if he wished, above all else, to be with them; but it is not for him.

The chief parental influence is the maternal relationship. She loves the child and the love is returned. There is increasing dependence, there is resentment, from which results an ambivalence of emotion which reacts adversely on the child. His insecurity is increased. He is out of step with his environment. There may be an incursion into a life of phantasy. The feelings of "out of touch with

reality" may be accentuated by school influences. His attempts at friendship are misunderstood. Vicious circles can occur, and the end result may be a deepening of the "stranger" feeling.

Dr. Meares deals separately with the introvert girl. The parents are less conscious of her deviation from normality, since she quietly plays with her dolls: "She will be a wonderful mother when she grows up." Traditionally boys should be extroverts and girls submissives. It is easy to see that the sex implications are different. In adolescence new problems arise, the boy is expected to "get a girl", the latter to be attractive to boys. Both are inadequate and react by various modes of evasion.

Adulthood does not solve the problem. Both the sexes feel insecure. Although they may outwardly conform to habits of social usage, there is a feeling of not fitting in with the environment. There is undue sensitivity, idealism and sexual inadequacy.

Dr. Meares discusses further problems which arise. The males tend to drift into student occupation, more particularly "teaching, psychology and theology"; employment in industry is unsatisfying, country life is distasteful. The female introvert tends to stay at home in order to keep her privacy.

Courtship presents new difficulties. The male may drift to prostitutes or homosexuality, the female to frigidity. Marriage may be disastrous. If the patient is lucky he may be saved by an intuitive wife.

The introvert's essential difficulty is emotional inadequacy. Treatment, whether by the doctor or by the parents, relatives or friends, must utilize a gradual emotional approach giving feelings of friendship and understanding. It can be illustrated thus:

The intuitive wife, by making emotional responses appropriate to his needs, establishes this close relationship with the introvert. He is now secure when he is with her. Now, and only now, she leads him out into an ever-widening circle of social contacts. This would have been impossible before, but she gives him security. He himself gradually becomes easier in these social contacts. As he becomes more secure, the intuitive wife gradually withdraws, and he is left standing on his own feet in a new-won freedom.

For the introvert wife, an understanding and patient husband is a necessity. Dr. Meares has skillfully woven the problem of our male and female introverts into a coherent pattern, at the same time indicating how others can help. This necessitates the telling of a long story to relatives and possibly friends. The time available to the medical practitioner, alas, often entails brevity. On such occasions advice to read "The Introvert" could be helpful. In this respect the volume has achieved its purpose and can be recommended.

**Diseases of the Liver and Biliary System.** By Sheila Sherlock, M.D., F.R.C.P., M.R.C.P.; Second Edition; 1958. Oxford: Blackwell Scientific Publications. 8½" x 5", pp. 736, with 213 illustrations. Price: 57s. 6d. (English).

IT is a pleasure to review the second edition of Sheila Sherlock's book. The framework has been extended to embrace advances in gastro-enterology which have followed the first edition. Chapters 1 to 4 cover diagnosis and laboratory procedures, including needle biopsy; Chapters 5 to 9 describe general manifestations of liver disease, including liver failure, portal hypertension and jaundice; the remainder describe specific diseases of the liver and biliary tract, including liver disorders in pregnancy and childhood. Free use of well chosen graphs, diagrams, photographs and occasional cartoons enlivens the text; comprehensive bibliographies follow all major sections.

The book illustrates well the advantages of single as against multiple authorship, for it is compact, non-repetitive and orderly in sequence. The stamp of Dr. Sherlock's original work appears throughout, particularly in sections on needle biopsy, portal venography, circulatory disease, metabolic disorders, and hepatocellular failure. For hepatic coma general supportive therapy with neomycin is now recommended, whilst glutamic acid, arginine and cortisone are all discounted.

The newer classification of cirrhosis may be viewed against some interesting statistical data relevant to aetiology. Of the author's cases of cirrhosis, 33% were presumed due to viral infection and 13% to alcoholic malnutrition, whereas in Australia as in America the proportion due to alcoholism is considerably higher. Almost half the author's patients had no ascertainable cause for their disease and were regarded as suffering from "cryptogenic cirrhosis". The elucidation of aetiological factors in this large group of cirrhotic patients constitutes an urgent and challenging problem. Until this is accomplished, classification of cirrhosis will rest on an unsure basis.

Other new material brings this edition completely abreast of present-day developments in liver disease. Modern views on bile-pigment metabolism, and the diagnostic value of transaminase estimations, splenic venography and intravenous cholangiography are now included. Moreover, the fascinating role of immunological disturbances in the perpetuation of hepatitis is discussed, and full recognition is made of the Australian work in this field. Corticosteroids are recommended for fulminant and lingering cases of virus hepatitis. References to the pancreas in the final chapters lead one to suggest that a chapter on pancreatic disease be included in subsequent editions.

"Diseases of the Liver and Biliary System" can be recommended to all physicians and surgeons without reservation, for it continues to be the best of several available text-books on liver disease.

**Regional Ileitis.** By Burrill B. Crohn, M.D. and Harry Yarnis, M.D.; Second Revised Edition; 1958. New York, London: Grune and Stratton. 8½" x 5", pp. 256, with 79 illustrations. Price: \$7.25.

MUCH discussion takes place still amongst pathologists and surgeons as to the causation and correct treatment of regional ileitis. It was with interest that we read the second revised edition of Crohn's book.

It is a beautifully produced little manual on the subject, covering every aspect of the disease. Opinions are substantiated by detailed analysis of many cases, or by many references to the literature. Unfortunately no finality has been reached concerning the nature of the disease or its treatment. This, one must assume, accounts for a baffling suspicion of vagueness, of reluctance to come to the point. Although the book is small, there is much padding. To take a small example, instead of a statement to the effect that economic status and race play no part in causation, a page is used to quote various opinions of no statistical significance whatever.

It is pleasing to note that it is easier in this edition to find out the author's views on surgical treatment, even though the case for enteroanastomosis as the best operation is not unassailably strong.

A contentious feature is the assumption that acute forms of jejunitis or ileitis are related to ileocolitis. Nor will all agree with the statement that ileitis and idiopathic ulcerative colitis constitute allied diseases.

**Medicine and Man: The Story of the Art and Science of Healing.** By Ritchie Calder, C.B.E.; 1958. London: George Allen and Unwin, Limited. 8½" x 5½", pp. 265. Price: 16s. (English).

THE publishers of this book have supplied very little information as to the medical background of its author, Ritchie Calder, although there is indirect evidence that he has written several popular works on subjects relating to general science or to world travel, and has been the recipient of a distinguished order for his services to the British Empire. Nevertheless, one gains the impression from occasional hints throughout the text that he is a qualified medical practitioner, with a philosophical outlook and a firm grasp of the scientific principles which have contributed to the evolution of modern medicine since the revival of learning towards the end of the fifteenth century.

The first part of the book is disappointing and not always easy to follow by reason of the long sequences of *staccato* sentences, the light-hearted references to witches, demigods and demons, facetious sallies at the expense of orthodox practitioners of a darker age and many undignified descents into the vernacular as a concession to less critical minds among the reading public. Hence, it came as somewhat of a surprise that the greater part of the book relating to advances made in the course of development of the allied sciences, with their influence upon enlightened concepts in the theory and practice of medicine, immediately revealed a dignified and fluent style with clarity of expression and scientific wisdom, giving the reader fresh insight into an extensive field of knowledge where the writer is evidently in his element.

In recent years quite a number of popular books have been written on exciting phases of medical history. Not a few of them have been compiled by authors who have no medical qualification, but who strive to atone for this deficiency by the exercise of a vivid imagination and an appeal to popular taste by a journalistic approach in regard to language and sensationalism, paying little heed to recognized authority or documentary evidence in statements of fact. However, there is much to be said for a book like "Medicine and Man", which contains accurate information for the benefit of laymen and medical practitioners, who will be the first to agree that medical literature has become so

voluminous that in this age of highly organized specialism many of us are unable to see the wood for the trees.

It would seem that Dr. Ritchie Calder's expert opinion and advice were appreciated by the World Health Organization, which sent him on frequent global expeditions for the furtherance of its specific objects in the interests of community health and the prevention of disease.

**"D. W. Winnicott, F.R.C.P. (Lond.): Collected Papers: Through Pediatrics to Psycho-Analysis"; 1958. London: Tavistock Publications, Limited. 8½" x 5½", pp. 360, with illustrations. Price: 35s. (English).**

THE content of this book is summarized in the preface. In the first section are reprinted Winnicott's views, written in 1931, on "normality and anxiety and fidgetiness in children" prior to his training in psychoanalysis.

The second section is a group of papers on psychoanalytically orientated paediatrics. The subjects discussed include appetite and emotional disorder, anxiety associated with insecurity, and child department consultations.

The final section, and the major part of the book, discusses the author's contributions to current psychoanalytical theory and practice. Some of the titles in this group are: "The Manic Defence", "Primitive Emotional Development", "Birth Memories", "Birth Trauma and Anxiety", "Withdrawal and Regression", "Psychosis and Child Care", and "The Depressive Position in Normal Emotional Development".

The author's long experience in paediatrics has assured not only a varied collection of clinical material but histories and theories written in a way that can be easily understood by any person interested in the psychological problems of children.

## Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

**"Electrocardiography",** by E. Grey Dimond, M.D.; Second Edition; 1958. Kansas: University of Kansas Medical Center. 11½" x 8½", pp. 174, with many illustrations. Price: \$4.85.

The syllabus for electrocardiography in the University of Kansas School of Medicine.

**"The Penguin Song Book",** by Leslie Woodgate; 1958. Mitcham: Penguin Books, Limited. 7½" x 5", pp. 192. Price: 7s.

A collection of old favourites with words and music.

**"Heredity of the Blood Groups",** by Alexander S. Wiener, A.B., M.D., F.R.C.P., F.C.A.P., and Irving B. Weisler, A.B., M.D., F.A.C.P.; 1958. New York and London: Grune and Stratton, Incorporated. 8½" x 5½", pp. 160, with 4 figures and 51 tables. Price: \$6.00.

The purpose of this monograph is to present in a unified manner the facts known about the subject.

**"A Doctor in Parliament",** by Donald McL. Johnson; 1958. London: Christopher Johnson. 8½" x 5½", pp. 288. Price: 18s.

The fourth volume of Dr. Johnson's autobiography.

**"Recent Advances in Paediatrics",** edited by Douglas Gairdner, M.A., D.M., F.R.C.P.; Second Edition; 1958. London: J. and A. Churchill, Limited. 8" x 5", pp. 388, with 82 illustrations. Price: 48s. (English).

Contains 13 chapters on specific subjects, all by experienced contributors.

**"So You Have Glaucoma",** by Everett R. Veirs, M.D.; 1958. New York and London: Grune and Stratton, Incorporated. 9" x 5½", pp. 70, with 10 illustrations. Price: \$2.75.

A book for the patient with glaucoma, intended to give him a better understanding of his condition.

**"The Year Book of Medicine (1958-1959 Year Book Series)",** edited by Paul B. Beeson, M.D., C. Muschenheim, M.D., W. B. Castle, M.D., T. R. Harrison, M.D., Franz J. Ingelfinger, M.D., P. K. Bondy, M.D.; 1958. Chicago: The Year Book Publishers, Incorporated. 7½" x 4½", pp. 784, with 125 illustrations. Price: \$7.60.

One of the Practical Medicine Series of Year Books.



## The Medical Journal of Australia

SATURDAY, JANUARY 17, 1959.

### A SIGNIFICANT DEVELOPMENT IN MEDICAL RESEARCH AND POST-GRADUATE WORK.

AN important statement relating to the Post-Graduate Medical Foundation of the University of Sydney is published in this issue (see page 88). This is commended to the attention of our readers, especially those in New South Wales, not only because it bears the names of the Chancellor, the Dean of the Faculty of Medicine and the Chairman of the Post-Graduate Committee in Medicine in the University of Sydney, but also because it embodies significant developments in medical research and post-graduate work in this country. The setting up of this Foundation is a major forward step, and fulfilment of its objects could be of great assistance to Australian medicine in its widest aspects.

It has long been recognized that teaching and research go hand in hand, and this is the pattern in most undergraduate schools of medicine. The objects of the Foundation bring the two together on the post-graduate level, and this could mean much to both. The most apparent object is to ensure that neither suffers from lack of financial backing or from wasteful inefficiency in organization. It is vital that adequate funds should be available for these purposes, and there is little doubt that considerable private sources remain to be tapped. At the same time, business firms and the like cannot be expected to make large sums available except to an efficient and sound organization. This the Foundation aims to be.

The objects of the Foundation are set out in the following terms in its constitution:

The objects of the Foundation shall be to assist the Senate of the University of Sydney and the Vice-Chancellor on matters associated with the promotion of Post-Graduate Education, Study, Work and Research in Medicine and the advancement of the Art and Science of Medicine, and in particular

- (a) to co-operate with the Post-Graduate Committee in Medicine and the Faculty of Medicine in the furtherance of Post-Graduate Education and Research in Medicine;
- (b) to support the Post-Graduate Committee in its public relations in the promotion of these objects;
- (c) to admit to membership of the Foundation, persons, firms, companies, institutions and associations, whether incorporated or unincorporated, and upon such terms and with such privileges as may be determined by the Foundation from time to time;
- (d) to enter into any arrangement with any institution or organization which has objects similar to those of the Foundation;

(e) to solicit donations, gifts and bequests to the University of Sydney from members of the public for the promotion of the objects of the Foundation; and

(f) to do all such things as are incidental or conducive to the attainment of the above objects or any of them.

These objects are wide. However, wisely and zealously pursued, they could do a great deal for medical research and post-graduate work in New South Wales and could set a pattern for Australia.

In relation to research there is no suggestion of any attempt to control the activities of research workers; this properly remains in the hands of the university departments and teaching hospitals concerned. The aim is to provide all material help and encouragement possible to enable the research to go on. Likewise, the furtherance of post-graduate work remains the responsibility of the Post-Graduate Committee, which also needs finance if it is to develop on imaginative lines a field of education which is still only in the experimental stage. The idea outlined in the statement of extending it, in coordinated programmes, to groups outside the medical profession is largely new, in this country at least, and exciting in its possibilities. What other developments will take place cannot yet be predicted. Certainly it seems time to put aside the notion that post-graduate education is merely an extension of undergraduate education and can profitably follow along the same lines. Perhaps it should develop in very different patterns.

The published statement points out that a public appeal for funds for the Foundation is to be launched on March 13 of this year. Members of the medical profession can help to prepare the ground for this by making the Foundation known to possible donors, large and small, and by encouraging in the community a sympathetic interest in medical research and post-graduate work. These are goods that can be sold with confidence. Doctors should also consider their personal responsibilities to the Foundation and help to give a lead. There is, of course, no need for anyone to wait for the public appeal to be launched. The Foundation is already active and in need of practical support.

### THE USE OF ABBREVIATIONS IN THE JOURNAL.

FOR many years no abbreviations of any kind were used in THE MEDICAL JOURNAL OF AUSTRALIA as a matter of firm editorial policy. More recently a few common abbreviations have crept in, but it has now been decided to fall into line with world-wide practice and to adopt standard abbreviations for such things as weights and measures. A list of these will be found on page 85, and separate copies of this list will be supplied on request. So far as possible, an accepted standard has been adopted, such as that of the British Pharmacopœia. For a few shorter words, such as gramme, grain and litre, the use of an abbreviation has been rejected; little would be gained by its adoption, and it was particularly wished to avoid confusion between grammes and grains in relation to dosage. It will be seen that the list is small, but it is probably sufficient so far as medical and scientific terms are concerned. For names of organisms, etc., we follow standard practice, with the

special provision that the name is printed in full at its first appearance in an article and its abbreviated form thereafter (e.g., *Staphylococcus aureus*, then *Staph. aureus*). Abbreviations of a more general character (U.S.A., C.B.E., B.M.A., R.A.A.M.C., etc., i.e., e.g., *ibid.*, and so on) are in a different category and do not lend themselves to the making of rigid rules. In general, if they are not ugly or obscure, they are acceptable. However, their use or otherwise in a particular context is a matter of somewhat arbitrary literary judgement, which will have to be left to editorial discretion. The form of abbreviations for names of journals in references and bibliographies is in process of being changed over to that of "World Medical Periodicals", published by the World Medical Association.

Because of the ordinary machinery of preparing material for publication, a period of overlap is inevitable in the adoption of these changes, but they will be brought into consistent use as soon as possible. We hope that, with other changes planned for the coming year, they will help to make the Journal more generally useful and acceptable.

## Current Comment.

### CUSHING'S SYNDROME AND ADRENALECTOMY.

In 1932 Cushing first clearly defined a rare syndrome which now bears his name, and which has since come to be recognized as one of the classical examples of endocrine dysfunction. This syndrome is caused by excessive secretion of adrenocortical hormones, principally hydrocortisone, and this in turn is due either to the presence of an adrenocortical tumour or, more often, to bilateral adrenocortical hyperplasia. In cases due to the presence of a unilateral tumour, treatment consists of removal of the tumour, though this is often malignant, and post-operative substitution therapy is frequently necessary owing to suppression of activity of the contralateral adrenal gland. Before the advent of cortisone and hydrocortisone, in cases in which the disease was due to bilateral adrenocortical hyperplasia it could sometimes be ameliorated by the removal of one adrenal gland, but the final outcome was almost invariably fatal, as the radical subtotal resection required to cure the condition was too dangerous in the absence of adequate hormone therapy. However, since hydrocortisone and cortisone have become available, a number of authors have described the successful treatment of bilateral adrenal hyperplasia by the removal of at least 80% of adrenal tissue, and in severe cases by total extirpation of both adrenal glands. The administration of cortisone must be begun before operation, to cover the sudden deprivation of cortical steroids by adrenalectomy, and must be continued for some time afterwards; when total adrenalectomy has been performed, permanent maintenance therapy will, of course, be required.

Two recent papers discuss the management of such cases in considerable detail. One by A. Stuart Mason, J. E. Richardson and C. E. King,<sup>1</sup> of the London Hospital, describes their experience in the treatment by adrenalectomy of Cushing's syndrome associated with bilateral adrenal hyperplasia, and outlines the therapeutic regime which they favour. Their data are drawn from records of 17 patients with adrenal hyperplasia, and one patient with Cushing's syndrome due to an adrenal carcinoma. The other is by two American workers, E. H. Ellison and G. D. Hamwi,<sup>2</sup> of Columbus, Ohio. In this paper the authors discuss hyperfunctioning lesions of the adrenal cortex in the light of previously reported cases and of 12 cases in which operation was performed at Ohio State University Hospital

since July, 1950. These 12 cases included eight of primary Cushing's syndrome, two of Cushing's syndrome secondary to a pituitary tumour, one of primary aldosteronism, and one of congenital virilism associated with an adrenocortical carcinoma.

In both papers the question of diagnosis is discussed. The English authors state that adrenalectomy should be undertaken only when the diagnosis of Cushing's syndrome has been proved beyond doubt. They point out that a form of benign transient endocrine dysfunction which may occur at puberty can be confused with Cushing's syndrome. They also state that in their view adrenalectomy is seldom justified in the adrenogenital syndrome in women, which, though it may be disfiguring, is not progressive or fatal. The diagnosis of Cushing's syndrome requires investigation of both the disorders of function and the anatomical lesion. Investigations of the latter may prove difficult. In many cases in which a tumour is present, this may be detected by straight X-ray examination and tomography combined with intravenous pyelography. If the presence of a tumour is suspected but cannot be proved by other means, the American authors suggest presacral insufflation of carbon dioxide and aortography as useful aids to diagnosis. However, the English team do not think that aortography is justifiable, as they regard the procedure as difficult and unreliable in obese subjects, and not without risk. They consider that, though it is advantageous to determine the presence and location of an adrenal tumour, it is not essential, and that the final decision about the anatomical lesion must be made at operation. In hyperplasia both adrenal glands are enlarged; if a functioning tumour is present, the opposite adrenal gland undergoes atrophy; therefore, they state, the type of lesion can be determined when the first adrenal gland is exposed.

Ellison and Hamwi state that in Cushing's disease both the plasma and the urinary levels of hydrocortisone are greatly increased, and the response of these levels to stimulation with corticotrophin can be used to distinguish cases in which the disease is caused by a tumour from those in which it is caused by bilateral hyperplasia. In the former case stimulation with corticotrophin has little or no effect, but in the latter there will be more than the usual threefold increase in the plasma level of hydrocortisone after a six-hour infusion of 25 milligrammes of corticotrophin; there will also be an appreciable increase in the amount of hydrocortisone in the urine.

In cases of bilateral adrenal hyperplasia, Mason, Richardson and King recommend that total adrenalectomy should be performed for younger patients and for those with rapidly progressive disease; for older patients they prefer subtotal adrenalectomy. In their younger patients they perform the operation in one stage and in older patients in two stages. Ellison and Hamwi regard severe hypertension as their sole criterion for total adrenalectomy, since in these cases the leaving of even a small remnant will result in persisting hypertension. For subtotal adrenalectomy the English team recommend the complete removal of the right adrenal gland, and the removal of all but one-eighth or one-tenth of the left; they argue that, if subtotal resection of each adrenal was performed, in case of relapse it would be impossible to decide which side should be reexplored; and they consider that hypertrophy of a remnant on the right side is more difficult to deal with owing to proximity of the vena cava. On the other hand, the American pair prefer to remove the whole of the left adrenal gland, leaving only sufficient of the right one to maintain optimum physiological activity. They emphasize that at operation the anterior transabdominal approach should be used, because the surgeon should be able (i) to visualize both the adrenal glands before deciding on his exact course of action, (ii) to explore the ovaries for "disease stimulating cortical hyperfunction or tumour" and (iii) to examine the sites of cortical rests for aberrant tissue. In contrast, the English team use a lateral approach, resecting the eleventh rib.

There are other points at which these two papers could be compared and contrasted. We have not ventured to embark on a discussion of the detailed metabolic studies

<sup>1</sup> *Lancet*, 1958, 2: 649 (September 27).

<sup>2</sup> *Arch. Surg.*, 1958, 76: 843 (June).



which make up the greater part of the English paper, or of the details of cortisone therapy, discussed by both, which is such a vital part of treatment both before and after operation. Ellison and Hamwi include in their paper a discussion of the other conditions to which adrenocortical hypersecretion may give rise. Two main disorders have long been recognized—Cushing's syndrome, caused by excessive secretion of hydrocortisone, and the adrenogenital syndrome, due to excessive production of adrenal androgens. To these has recently been added aldosteronism, caused by over-production of one of the latest additions to the known members of the hormone family, aldosterone. Each of these anomalies may result either from an adrenocortical tumour or from adrenocortical hyperplasia, and elements of the different syndromes are frequently combined. Ellison and Hamwi's case of primary aldosteronism associated with adrenocortical hyperplasia is one of the very few yet described.

The treatment of this disease illustrates well some of the most stimulating aspects of modern medicine. It presents a situation which calls for both a high degree of specialization and good teamwork between widely different disciplines. The two papers quoted also provide an interesting example of the way in which two highly expert teams, tackling the same problem with the benefit of all the latest advances in their subject, yet differ in significant points of emphasis and technique, showing how diversity may ultimately lead to the best possible final agreement.

#### THE HARVEY TERCENTENARY CONGRESS.

On June 3, 1957, a congress was opened at the Royal College of Surgeons, London, to commemorate the tercentenary of the death of William Harvey. Biologists, physicians and surgeons from all over the world attended to pay homage to the memory of one of the greater figures in medical history. When, in 1628, Harvey published his *De Motu Cordis et Sanguinis*, in which he detailed his experiments and deductions to prove that there was a circulation of the blood, he produced a document that was to revolutionize the study of physiology and form the basis for all modern advances in medicine. Later in life he was to write his *De Generatione*, so full of his deep knowledge of embryology and his fundamental discoveries in that field. Rather naturally his work on the circulation far eclipsed his embryological researches, for his deductions were so much against the current teaching and so original, and yet at the same time so clearly proved by experiment, that they resulted in widespread controversy and even vituperative argument before receiving general acceptance. It was only to be expected, therefore, that when this tercentenary congress was planned, the theme was to be the circulation; history was not forgotten, but the emphasis was to be on the modern approach to the study of the heart and circulation.

The proceedings of the congress have now been published, ably edited by Professor John McMichael of London.<sup>1</sup> The volume is not only a record of a highly successful meeting, but a document of importance, for here is set down a great number of facets of modern research on the heart and circulation, together with a review of much of our present knowledge of the fascinating and complex problems involved in this study. An appreciation of Harvey's life and work by a number of historians forms a brief, perhaps too brief, introduction to the main portion of the proceedings. It is not possible here to give an account of the 30 or so papers on modern research. All are by authorities from many countries. Many describe methods of experimental research that were unheard of 30 years ago. It is fascinating to wonder what Harvey would have thought of the modern developments of his simple but well-planned experiments, as well as wondering what

Harvey's reactions would have been to colour television and to the trans-Atlantic radio-telephone discussion that took place at the congress. One feels sure that he would have been amazed and even somewhat disconcerted, but he would certainly have been keenly interested and would have approved of what he saw.

These proceedings form a unique tribute to the memory of William Harvey. Indeed, they are the culmination of a number of tributes, for tercentenary commemorations were held in many parts of the world. The London congress was certainly the largest and most brilliant of them all. Many of us have seen the magnificent colour film produced for the Royal College of Physicians which formed merely a part of the congress. Mr. Dickson Wright, the President, his committees and all the many others who helped towards the success of the meeting deserve our congratulations on a job well done. Professor McMichael has our gratitude for his painstaking work in producing a permanent record of the congress.

#### STANDARD ABBREVIATIONS AS USED IN "THE MEDICAL JOURNAL OF AUSTRALIA".

Ångström unit	.. . . .	Å
Bacille Calmette-Guérin	.. . . .	B.C.G.
Blood pressure of 175 millimetres of mercury, systolic, and 90 millimetres, diastolic	.. . . .	175/90 mm. of mercury
Centigrade	.. . . .	C.
Centimetre(s)	.. . . .	cm.
Cubic millimetre(s)	.. . . .	c.mm.
Cubic micron(s)	.. . . .	c.μ
Dose, A, lethal to 50% of the sample	.. . . .	LD <sub>50</sub>
Drachm(s)	.. . . .	dr.
Fahrenheit	.. . . .	F.
Fluid ounce(s)	.. . . .	fl. oz.
Gramme (not abbreviated)	.. . . .	g.
Grain (not abbreviated)	.. . . .	gr.
Inch(es)	.. . . .	in.
International unit(s)	.. . . .	I.U.
Kilogram(s)	.. . . .	kg.
Litre (not abbreviated)	.. . . .	l.
Mean corpuscular hæmoglobin	.. . . .	M.C.H.
Mean corpuscular hæmoglobin concentration	.. . . .	M.C.H.C.
Mean corpuscular volume	.. . . .	M.C.V.
Microcurie(s)	.. . . .	μc.
Microgramme(s)	.. . . .	μg., γ
Micromicrogramme(s)	.. . . .	γγ
Micron(s)	.. . . .	μ
Millicurie(s)	.. . . .	mc.
Milliequivalents per litre	.. . . .	mEq/l.
Milligramme(s)	.. . . .	mg.
Millilitre(s)	.. . . .	ml.
Millimetre(s)	.. . . .	mm.
Millimicron(s)	.. . . .	mμ
Minim(s)	.. . . .	min.
Minimum lethal dose	.. . . .	M.L.D.
Number	.. . . .	No.
Ounce(s)	.. . . .	oz.
Parts per million	.. . . .	p.p.m.
Pound(s)	.. . . .	lb.
Revolutions per minute	.. . . .	r.p.m.
Röntgen unit(s)	.. . . .	r.
Standard deviation	.. . . .	S.D.
Volume in volume	.. . . .	v/v
Volume in weight	.. . . .	v/w
Weight in volume	.. . . .	w/v
Weight in weight	.. . . .	w/w

NOTE.—Abbreviations of units of measurement are normally used after numerals, but not otherwise. For example: "12 cm.", but "measured in centimetres".

<sup>1</sup> "Circulation: Proceedings of the Harvey Tercentenary Congress", edited by John McMichael, M.D., F.R.C.P., F.R.S.; 1958. Oxford: Blackwell Scientific Publications. 32" x 5½", pp. 538, with many illustrations. Price: 50s. (English).

## Abstracts from Medical Literature.

### SURGERY.

#### Lesions of the Salivary Glands.

D. G. McEACHEN *et alii* (*Surg. Gynec. Obstet.*, June, 1958) review 155 cases of salivary gland lesions seen in the Saskatoon Cancer Clinic from 1932 to 1956. One hundred and twenty-nine were neoplasms. Seventy-nine of these neoplasms were benign mixed tumours. Recurrence rates and five-year survival rates in patients free of disease are recorded. Benign mixed salivary tumours have an over-all recurrence rate of 6%. Patients with malignant tumours have an over-all five-year survival rate of 65%. In general, the treatment was surgery plus post-operative irradiation. The details of the radiation technique and dosage are stated. Evidence is produced to show that salivary tumours do respond to adequate irradiation and that the best results will be obtained with adequate surgery plus adequate irradiation.

#### Hæmorrhage from Oesophageal Varices.

E. L. MEINE AND L. J. WITKOWSKI (*Amer. J. Surg.*, July, 1958) discuss the problem of acute bleeding from oesophageal varices in 42 patients seen between 1950 and 1955. No patients admitted for medical treatment or elective surgery because of remote bleeding were included. Presenting symptoms were hæmatemesis or melena, or both. Of 25 patients with known liver disease, 23 had had previous hæmorrhages. Physical findings included, in order of frequency, hepatomegaly, splenomegaly, jaundice and ascites. Portal cirrhosis was the most frequently found underlying liver disease. The history revealed alcoholism in 12 of the 33 patients. A Sengstaken tube was used on seven patients. After acute bleeding, 14 patients underwent 16 surgical procedures, ranging from exploration to major resections and shunt procedures. There were five post-operative deaths and six patients died without surgical intervention. There were two post-operative deaths among ten patients who underwent a porta-caval shunt operation. While surgical procedures which reduce portal hypertension appear to prolong life in some patients, the prognosis generally is not good after hæmorrhage from oesophageal varices.

#### Coronary Heart Disease.

C. S. BECK (*Amer. J. Surg.*, May, 1958) discusses the causes of death in coronary artery disease, basing his observations on experience of 6000 experimental operations on dogs and 500 operations on patients with the disease. The three dominant causes are (i) the production of an unstable electrical condition in the heart, (ii) the reduction of total coronary artery inflow, (iii) the destruction of muscle. In most instances there is one dominant factor with one or both other factors contributing. Uniformity of oxygenation in the heart muscle is accompanied by electrical stability in the

heart whether the heart is well oxygenated or cyanosed. An uneven distribution of oxygen is accompanied by electrical instability due to currents between pink and blue muscle. These currents can produce anginal pain, fibrillation and death. In these cases the total inflow of blood is adequate but the distribution is uneven. The aim of treatment is the production of intercoronary arterial communications; these can be produced by surgical operation. The coronary arteries may be so severely stenosed that the amount of blood delivered to the heart is insufficient to sustain the beat. If the distribution of inflow is uniform, owing to the development of intercoronary communications, fibrillating currents are less likely to form and the stenosis can become so severe that the muscle loses its power of contractility although the muscle remains viable. The incidence of this type of death is probably not high, because the stenosing process usually ends with fibrillating currents. Treatment of reduction of total inflow requires more blood. One method is by grafting various tissues upon the heart; vessels large enough to transfer blood to the heart do not develop, and the value of the procedure lies in the inflammatory reaction which helps develop the intercoronary channels. Another method investigated is conversion of the coronary sinus into an arterial inlet. Intimal thickening of the veins blocks the flow after some weeks, but meanwhile intercoronaries may develop. A direct approach on the coronary arteries has nothing to offer. The third main cause of death, destruction of muscle, occurs when infarction of larger or smaller areas proceeds without producing electrical currents of fibrillating capacity. The total coronary inflow is adequate to support the heart, but there is not enough contractile muscle left to circulate the blood. The treatment of this condition is the same as that of myocardial failure. Replacement of scarred muscle is not possible.

#### Sphincteroplasty for Recurrent Pancreatitis.

S. A. JONES, L. L. SMITH AND G. GREGORY (*Ann. Surg.*, February, 1958) discuss the rationale and technique of transduodenal sphincteroplasty, and the results of this operation in 28 cases of recurrent pancreatitis. They postulate that pancreatitis may result from a combination of stimulation of the gland with intra-pancreatic or extra-pancreatic duct obstruction. Although the presence of extra-pancreatic duct obstruction is frequently not demonstrable in patients with recurrent pancreatitis at operation or autopsy, it could still be explained by contraction of the duodenal musculature. Anatomical studies in 50 dissections appeared to show that contraction of the duodenal musculature could cause compression of both the common bile duct and the pancreatic duct. Moreover, destruction of the fibres encircling the common duct in its intramural course will disrupt the contracting mechanism which surrounds the pancreatic duct, since the fibres between the two ducts are few. The object of sphincteroplasty is not only to destroy the sphincter function at the outlet of the common duct, but also to destroy the constricting action of

the duodenal wall muscle fibres on the pancreatic duct. Obviously the operation would not benefit patients with pancreatitis due to intrapancreatic duct obstruction, and at operation a retrograde pancreatogram should be obtained to exclude this possibility. Common duct pressure studies after simple common duct exploration or sphincterotomy showed a slow rise after an intravenous injection of morphine. After sphincteroplasty there was a rapid and short-lived rise in pressure followed by a fall to the base-line. Post-operative cholangiograms after an intravenous injection of morphine demonstrate duodenal wall contraction, with resulting obstruction of the common duct in patients after sphincterotomy. In the sphincteroplasty group morphine produced no effect. It is considered that pressure changes within the pancreatic duct will in some degree run parallel with those in the common duct. Twenty-five of the 28 patients were adequately followed up. There was no recurrence of pancreatitis after the operation in 19 cases, a solitary attack in three cases, more than one attack but the patient much improved in two cases, and no improvement in one case.

#### Heterotopic Pancreatic Tissue Involving the Stomach.

N. S. MARTINEZ *et alii* (*Ann. Surg.*, January, 1958) report on 51 surgical cases of heterotopic pancreatic tissue involving the wall of the stomach recorded in the files of the Mayo Clinic between 1907 and 1954 inclusive. The lesions were situated almost exclusively in the pyloric and prepyloric regions, and were mostly submucosal, producing a sessile, polypoid, or nipple-like umbilicated mass. Ulceration of the overlying mucosa, hæmorrhage, mechanical obstruction and neoplastic change sometimes occur. The condition is often symptomless, but 28 patients complained of symptoms indistinguishable from those produced by disorders of the stomach, duodenum, or gall-bladder. In 74% of the cases a barium meal X-ray examination gave positive findings, which were most frequently consistent with those of a polypoid tumour. Occasionally a radiological diagnosis of heterotopic pancreatic tissue is justified by the demonstration of barium-filled ducts within a small filling defect in the antrum. To confirm the diagnosis and exclude malignancy, surgical excision becomes necessary because of the presence of symptoms and radiological signs.

#### Amylase Concentration in the Diagnosis of Acute Pancreatitis.

J. R. AMERSON, J. M. HOWARD AND K. D. J. VOWLES (*Ann. Surg.*, February, 1958) report studies of the amylase concentration in serum and peritoneal fluid after acute perforation of gastroduodenal ulcers. They emphasize that in acute pancreatitis a successful non-operative regimen depends on accurate diagnosis, so that no patient with a disease for which operation is life-saving is erroneously diagnosed as having acute pancreatitis. Confusion may arise because acute perforation of a gastric or duodenal ulcer may produce a striking rise in serum amylase concentration. In 1949 studies of the peritoneal fluid obtained



at laparotomy from patients with acute pancreatitis revealed very high concentrations of pancreatic enzymes. The next step appeared to be the determination of amylase concentrations in the peritoneal fluid as well as serum in patients with acute perforation of gastro-duodenal ulcers. Out of 41 such patients the serum amylase level was significantly elevated in nine. In 26 in whom the amylase concentration of the peritoneal fluid was measured, the concentration was above 200 Somogyi units in 17 and above 1500 units in three. It is concluded that there is no concentration of the enzyme in serum or peritoneal fluid that is diagnostic of acute pancreatitis; however, these investigations may often prove a useful adjunct to clinical evaluation.

### The Pathogenesis of Pancreatic Necrosis.

L. C. REID *et alii* (*Surgery*, April, 1958) discuss the mechanism of the pathogenesis of pancreatic necrosis and the therapeutic effect of propylthiouracil. They present evidence to suggest that the mechanism of necrosis of pancreatitis is the result of a vascular lesion with loss of cell membrane continuity, and the escape of intracellular enzymes which destroy all inadequately irrigated pancreatic or other organ cells with which they come in contact. The authors state that trypsin and chymotrypsin play no significant role in this process. Propylthiouracil inhibits oxidative energy release, less work is performed, less product is formed, less supplies are consumed, and as a result the gland is placed at rest when under stress. The authors point out that in view of the enormous destructive capacities of the intracellular pancreatic enzymes, the maintenance of the integrity of the cells becomes of the utmost importance. Cutting down of the synthesizing activities of the gland should allow more materials and energy to be available to maintain cell structure in the presence of restricted supplies because of some vascular derangement. Accordingly, the authors state that propylthiouracil should be clinically beneficial in the delay and limitation of the necrotic process. They state that this beneficial effect has been demonstrated.

### Papillary Carcinoma of the Thyroid.

C. R. UNDERWOOD, L. V. ACKERMAN AND C. ECKERT (*Surgery*, April, 1958) present a study of 62 patients with papillary carcinoma of the thyroid and discuss the surgical treatment of the condition. In this series the usual presenting symptom was a nodule in the thyroid with or without enlarged lymph nodes. In some cases lymph node enlargement alone was the presenting symptom. Thyrotoxicosis appeared to have no significant relationship to this type of tumour. There was no correlation between minute variations in microscopic pathology and prognosis. Local removal of the tumour was frequently followed by local recurrence. The authors conclude that papillary carcinoma of the thyroid is a potentially lethal tumour. They make the following recommendations as regards treatment: (i) When operating upon a lesion of the thyroid gland,

suspected of being a neoplasm, the surgeon should resect the entire involved lobe by extracapsular dissection. If the tumour involves the isthmus or the opposite lobe, then a wide margin should be taken about the area of extension. Should the growth be invading contiguous structures of the neck, then each case must be dealt with on its merits. Various possible situations are discussed. (ii) Should a diagnosis of papillary carcinoma be made, a subtotal resection of the opposite lobe leaving only a small amount of normal glandular tissue on the posterior capsule should be performed. Great care must be taken to preserve viable parathyroid gland and the recurrent nerve must be carefully identified. The authors state that the effects of bilateral total lobectomy do not justify this procedure in dealing with papillary carcinoma, unless involvement of both lobes is massive. (iii) In the presence of palpable lymph-node metastases, a definitive neck dissection should be performed. As such palpable metastases are most commonly encountered beneath the sterno-mastoid muscle, this will mean a radical neck dissection with sacrifice of the sterno-mastoid muscle and internal jugular vein. In addition, the authors routinely remove the submaxillary lymph nodes. (iv) After the diagnosis of metastatic papillary carcinoma by excision of a node in the neck, in the absence of a clinically obvious tumour in the thyroid, a total lobectomy should be performed on that side and a subtotal lobectomy on the contralateral side, with a radical neck dissection on the side with demonstrable metastases. The incision through which the biopsy specimen was obtained should be included in the en-bloc resection. (v) If it subsequently becomes apparent that disease persists in the neck, further surgical removal may still control the growth, especially when the persistence is in a lymph node. Irremovable or metastatic lesions should be treated with thyroid hormone in dosages of 180 to 300 milligrammes per day. X-ray therapy may control the pain from bony metastases and promote recalcification and healing in pathological fractures. In the authors' experience, radioactive iodine has been of little value in the treatment of papillary carcinoma.

### Effect of Unilateral Radical Neck Dissection on Intracranial Pressure.

H. M. MORFITT AND H. CLEVELAND (*A.M.A. Arch. Surg.*, May, 1958) point out that the operation of radical neck dissection, which involves sacrifice of the internal jugular venous system, usually on one side, is the most effective way of dealing with metastatic cervical carcinoma. They state that no case of a permanent increase in intracranial pressure resulting from unilateral sacrifice of this portion of the return blood flow from the brain has ever been published. Many observers have noted a transient rise in intracranial pressure, but compensation has always occurred as soon as the collateral pathways have had an opportunity to come into play. The authors, however, report two cases in which an increase in intracranial pressure after removal of a single internal jugular venous system developed to a degree

sufficient to threaten total blindness. Initially they suspected that such intracranial complications were probably due to an unrecognized intracranial metastasis, but a follow-up over a period of five years and eight months respectively, with no additional evidence of an intracranial lesion, shows that this was not the cause of the increased intracranial pressure. This pressure has had to be compensated for by a subtemporal decompression in each instance. The authors consider that the anatomical basis for such a phenomenon is probably some rare lack of development of specific intracranial venous pathways, which makes it impossible for such patients to compensate by collateral circulation for the removal of one internal jugular venous system.

### Isolated Traumatic Rupture of the Ventricular Septum.

E. C. PEIRCE, C. H. DABBS AND F. L. RAWSON (*A.M.A. Arch. Surg.*, July, 1958) discuss the case of a 19 years old youth who sustained a rupture of his ventricular septum in a car accident. The injury was non-penetrating, the only chest fracture being a fracture of the left first rib posteriorly. The diagnosis was made seven days after the accident by finding the characteristic murmur and thrill, by the appearances of pulmonary congestion in the X-ray film, and by the electrocardiographic finding of a left bundle branch block. As his condition deteriorated, direct repair of the defect was carried out under hypothermia. The authors consider that this surgery was life-saving, although the closure did not remain complete. They point out that this is the eighteenth reported case of isolated traumatic ventricular septal defect, and they believe that this is the second case in which the lesion has been surgically repaired. They recommend a more aggressive approach to cardiac trauma.

### Pre-Operative Evaluation in Cases of Bronchogenic Carcinoma.

T. W. SHIELDS AND E. SHOCKET (*A.M.A. Arch. Surg.*, May, 1958) performed biopsies on non-palpable scalene lymph nodes in 47 patients with clinically resectable carcinomata of the lung and showed that three disclosed metastatic disease. As a result of this they conclude that the procedure does not have a sufficiently high yield of positive results to justify its routine use in all clinically resectable carcinomata of the lung. They advise its employment pre-operatively for those patients with a prominent "hilar mass" in the X-ray films of the chest, or when the bronchoscopic biopsy tissue or cytological smear reveals the presence of an undifferentiated carcinoma.

### Biopsy of the Pericardium and Myocardium.

M. WEINBERG, E. H. FELL AND J. LYNFIELD (*A.M.A. Arch. Surg.*, May, 1958) describe a technique for biopsy of the pericardium and myocardium under local anaesthesia which they have employed in five cases. They point out that surgical biopsy will add to our understanding of an important group of diseases about which much too little is known at present.

## University Intelligence.

### THE POST-GRADUATE MEDICAL FOUNDATION OF THE UNIVERSITY OF SYDNEY.

THE Chancellor of the University of Sydney, Sir Charles Bickerton Blackburn, the Dean of the Faculty of Medicine, Professor Bruce Mayes, and the Chairman of the Post-Graduate Committee in Medicine, Dr. V. M. Coppleson, have announced details of the Post-Graduate Medical Foundation of the University of Sydney and asked for the support of the medical profession and medical organizations. Their statement is as follows.

The Foundation was established by the Senate of the University of Sydney on July 7, 1958, on the recommendation of the Post-Graduate Committee in Medicine. Its objects, as set out in the Constitution, are "to assist the Senate of the University and the Vice-Chancellor on matters associated with the promotion of Post-Graduate Education, Study, Work and Research in Medicine and the advancement of the Art and Science of Medicine" and, in particular, "to co-operate with the Post-Graduate Committee and the Faculty of Medicine of the University in the furtherance of Post-Graduate Education and Research in Medicine" and "to support the Post-Graduate Committee in its public relations in the promotion of these objects".

As the result of meetings and discussions which have taken place since the establishment of the Foundation, the steps which will be necessary to translate these words into action are becoming clear.

The first objective will be to obtain finance and establish a central fund to assist the Faculty of Medicine, the Post-Graduate Committee and the Sydney teaching hospitals in the development of research and the provision of fellowships and scholarships.

How urgent is the need for support of medical research in the University and the teaching hospitals is underlined by the fact that, apart from research funds channelled into a few special objectives, the annual amount available for general medical research from the University's own funds in all its departments at present available does not exceed £5500, to which £2500 is added by the National Health and Medical Research Council, making a total of £8000. The teaching hospitals are also being restricted in their research activities by a similar lack of funds.

Unlike most other foundations and funds, in the scope of its assistance to research, the Post-Graduate Medical Foundation is not limited to one institution, hospital or department. Its funds will not be channelled into one aspect of medicine, but all fundamental and applied medical research and all medicine will be its field.

Applications have already been received by the Foundation from University departments and the teaching hospitals for assistance to general research for sums greatly in excess of those at present available. These requests cover research in cancer, cardio-pulmonary research, heart disease, chest diseases, prematurity, sterility, gastric and duodenal ulceration, radiotherapy, pathology, neurology, skin diseases and drowning.

The assistance asked for includes the setting up of isotope and other laboratories, special units, special museums, teaching exhibits, special research equipment, instruments and technical staff.

Funds are particularly needed to provide fellowships and scholarships for young men to work and train in research in the University departments and teaching hospitals. Included particularly in this group are many highly trained young men who, on their return to Australia after years of study and training abroad, can find no place to continue their studies and research.

Exchange of ideas is also of prime importance to medicine in Australia. This is another aspect of the function of the Foundation for which the Post-Graduate Committee and the Faculty of Medicine will look for special assistance. The isolation of Australia from the great world centres of medical education and research makes an active programme for the continual importation of new ideas and techniques imperative.

The traffic is two-way. Funds will be required to provide fellowships and scholarships for young and energetic men

to work and train in leading institutions abroad, so as to acquire new ideas and return with new methods and techniques.

Already the Foundation, with the meagre funds at its disposal, has provided assistance to enable Australian doctors to work with Professor Herbert Olivecrona in Sweden, with Professor Howard Florey at Oxford, and in the research departments of the Mayo Clinic. Twelve to fifteen fellowships each year at home and abroad are badly needed, and exchanges at a more senior level are also highly desirable.

The Foundation will also assist the Post-Graduate Committee and the Faculty in respect to the visits of eminent teachers from overseas, who will be invited to lecture, teach and work in the departments of our universities and teaching hospitals and, wherever possible, to visit all States of Australia and New Zealand. This has already been well developed by the Post-Graduate Committee and other post-graduate organizations of Australia. It has proved the medium of important contributions to medicine in Australia and contributed in no small way to the maintenance of the high standards of Australian medicine.

The list of visitors from abroad has included such famous names in medicine as Professor Charles Best, one of the discoverers of insulin, Sir Geoffrey Jefferson, Professor Herbert Olivecrona, Dr. George Pack, Dr. W. S. C. Copeman, Dr. Michael E. De Bakey and others, many of whom have left a permanent mark on medicine in Australia.

Not only is the Foundation unique in these respects as a university medical organization, but it breaks new ground by introducing a new concept into post-graduate medical education. This is to extend post-graduate medical education beyond its present limits so as to embrace not only the education of medical practitioners, but in certain instances other groups outside the medical profession, the non-medical aspects of which will be the responsibility of the Foundation.

This will mean that, in addition to professional programmes for medical men, concurrent programmes in certain instances will be specially arranged for para-medical groups, such as physiotherapists, nurses, social workers and technicians, and other programmes for community groups, specially informed lay groups, parents and others.

This is the technique which was followed with great success in the case of the visit of Dr. Ludwig Guttmann. In certain subjects, particularly in relation to the visits of special overseas visitors during the present year, this new plan will immediately be put into operation.

The first subject to be treated in this manner will be the subnormal and physically handicapped child. One of the first approaches and donations received on behalf of the Post-Graduate Medical Foundation was from the Watt Street Hospital Handicapped Children's Welfare Association, with a request for assistance. An equal sum was added to this by the Post-Graduate Committee from moneys provided by the Government of New South Wales.

An invitation was sent to Dr. L. T. Hilliard, the Superintendent of the Fountain Hospital, London, and a recognized authority on the subject, and he will visit Australia early in April, 1959.

After consultation with the Department of Mental Health, the Professor of Psychiatry, the New South Wales Mental Health Association, the Watt Street Hospital Handicapped Children's Welfare Association and other organizations for subnormal and physically handicapped children, a medical educational programme in Sydney and Newcastle has been arranged which will include a week's full-time post-graduate course at Newcastle, talks to social workers and physiotherapists, and discussions with the Child Welfare Department and various organizations. Talks will also be arranged by the Foundation to parents and friends in Sydney and Newcastle. The opportunity will at the same time be taken by the Foundation to state publicly the case of the subnormal and physically handicapped child in the Press, over the radio and on television. It is thus hoped to make Dr. Hilliard's visit the stimulus for an intensive study of these problems in Australia and to lay the foundation for further research into them.

Similar techniques will be adopted in respect to the visit about October or November, 1959, by Dr. L. G. Norman, Chief Medical Officer of the London Transport Executive and a member of the Expert Advisory Panel on Occupational Health of the World Health Organization, when the whole problem of traffic accidents will come under review, in both its lay and its medical aspects. Dr. Norman and Dr. Hilliard will also visit other States and New Zealand.



Later in the year, Professor Jethro Gough of the Welsh National University will talk to doctors on industrial and allied lung diseases and, in addition, will meet and discuss these problems with industrial and other organizations in Sydney, Newcastle, Wollongong and Broken Hill, as well as in other parts of Australia.

Advantage will also be taken of the visit of the Sims Travelling Professor, Mr. W. Gissane, to arrange lectures to police, industrialists, first-aid organizations and others in the latest methods of handling casualties, and a variation of the technique will be used for the visit of Dr. John J. Conley of the Pack Medical Group, New York, a leading authority on cancer of the head and neck, who will visit Australia in July.

The Foundation can also be expected to assist the Post-Graduate Committee and the Faculty of Medicine in the provision of teaching facilities, the establishment of a post-graduate school, the provision of hostels for post-graduate students and assistance to medical conferences, and in other ways. The Foundation has already promised to support a conference on radiobiology in Sydney during 1960. Assistance will also be provided on the para-medical and lay side. The Foundation, for instance, has already taken an interest in drowning and artificial respiration. It arranged with the Surf Life Saving Association of Australia to hold a special public meeting on artificial respiration addressed by Dr. A. C. Ivy, and has purchased and made available a recent film on the new and much-discussed method of mouth-to-mouth resuscitation. In all these aspects, the Foundation is capable of performing great national service, and its influence will obviously extend throughout Australia and New Zealand.

The method of financing the Foundation will primarily be "by admitting to Membership of the Foundation persons, firms, companies, institutions or associations, incorporated or unincorporated, as Governors, Members or Associate Members".

Large firms and organizations, who are the target for continual bombardment by applications for research grants, by contributing to the Foundation can direct such applications to the Foundation, where they can rest assured they will receive examination by an expert body.

The Foundation also offers an opportunity to leading firms, organizations and others to participate in its management, and in most instances where they subscribe the yearly sum of £1500 or more and nominate a governor, he may be invited by the Senate of the University to become a member of the Council of the Foundation. In this way the Foundation hopes for obtain the personal guidance and the active support of some of the ablest business executives and leaders of industry in New South Wales.

Already Australian Consolidated Press Ltd., Parke, Davis and Company Ltd. and Pfizer Pty. Ltd. have become governors of the Foundation, and their nominees, who are their managing directors, have been appointed by the Senate as members of the Council. Contributors of yearly sums of £500 and £250 will become members and associate members. Others have sent contributions to the Foundation.

In addition to enlisting governors and members, the Foundation will also solicit public donations, gifts and bequests, and will offer an opportunity to the public to subscribe in an appeal which will be opened on Friday, March 13, 1959.

Persons desiring to assist medical research are often unaware of how to proceed or may not wish to subscribe the sums necessary to become governors or members. To these and others wishing to make direct donations or gifts or bequests in their wills the Post-Graduate Medical Foundation offers the best opportunity to ensure that their donations are directed on expert advice to the right quarter and used to the best advantage.

All contributions and bequests to the Foundation are gifts to the University of Sydney and are free of all taxation and gift duty. The headquarters of the Foundation has been established at 131 Macquarie Street, Sydney.

Although no public appeal has yet been made by the Foundation, it has already received voluntarily, from public spirited persons and organizations who have been aware of its formation, over £10,000, much of which represents yearly contributions.

To achieve its objects it is estimated that the Foundation will require not less than £100,000 per annum. A copy of the constitution of the Foundation, together with financial and other details, can be obtained on application to the

Foundation. The members of the Council of the Foundation are Mr. Frank Packer (President), Mr. L. W. Farnsworth (Chairman), Sir Charles Blackburn, Major-General I. Dougherty, Professor S. H. Roberts, Professor W. M. O'Neill, Professor Bruce Mayes, Dr. V. M. Coppleson, Dr. George Bell, Sir Garfield Barwick, Mr. G. E. Rodney Brown, Mr. S. R. I. Clark, Mr. J. G. Cooper, Mr. R. Crichton-Brown, Mr. Vincent Fairfax, Mr. H. F. Giblin, Mr. W. L. Murray Robson and Mr. T. J. White.

It is our opinion, as Chancellor of the University, the Dean of the Faculty of Medicine and Chairman of the Post-Graduate Committee, that no body is better designed to raise the standards of medicine and medical research in our midst or more worthy of support.

## Out of the Past.

*In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.*

### THE PRINCE ALFRED HOSPITAL (1882).<sup>1</sup>

[From the Australasian Medical Gazette, December, 1882.]

The Alfred Memorial Hospital is now in working order, and nearly a hundred beds are in actual occupation. The same mastery of detail which characterises every structural item in this building is discernible in the professional and economic arrangements of the institution. The directors wisely began by appointing a "medical superintendent", a functionary long promised, but still desiderated in the older hospital in Macquarie Street. Attention to the medical requirements of the Alfred Hospital is provided for through a medical board, at which only members of the honorary staff have a seat—a day or two before the monthly meeting of the directors. The recommendations of this board are carried to the directors at their sitting the following day by its medical representative, and by this arrangement are ensured the deference they are entitled to. The plan works admirably. The principal subject of discussion at the last meeting was the distribution of medical duties. It was decided that the ordinary official rule of the London College of Physicians should apply here in connection with medical costs: that physicians should not be permitted to operate, and that a distinct department, with a special medical officer, should be organized for the treatment of diseases of women. It will, however, be essential for its success that the gentleman in charge shall have had abundant opportunities in Europe or America for familiarising himself with the thousand and one resources in this comparatively recent branch of the medical vocation. The complete design of the Alfred Hospital provides for 500 beds. It is certain that the attempts to carry this out through the medium of State funds will meet with powerful opposition.

## Correspondence.

### AUSTRALIAN RHEUMATISM ASSOCIATION (VICTORIAN BRANCH).

SIR: As a member of the Victorian Branch of the Australian Rheumatism Association, I wish to state that there appears to be a misunderstanding in reference to recent comments of Dr. Michael Kelly as published in THE MEDICAL JOURNAL OF AUSTRALIA of December 27, 1958.

Whilst sharing his anxiety *re* the apparent lethargy of the Australian Rheumatism Association, I must indicate that I received an agenda of the annual general meeting held at the Royal Prince Alfred Hospital, Sydney, on November 15, 1958, and preceded by a council meeting on November 12, 1958. The meeting for 1958 was to have been held in Melbourne. It is certainly disappointing to find that the original enthusiasm of 1956 has waned. Considering that more than 10% of the increasing Australian population is affected with some form of rheumatic disease, there is so much work to be done and, alas, so little time to do it in!

<sup>1</sup> From the original in the Mitchell Library, Sydney.

Admittedly rheumatology, physical medicine and rehabilitation in Australia are ten years behind the times when compared with world standards. Many of our colleagues would, however, be loath to make such an adverse admission. The cold fact remains that in only a few Australian medical schools is there room in the curriculum for training in these subjects, necessitating post-graduate experience abroad. And yet rheumatologists are endeavouring to face up to a difficult situation. The late Dr. L. Parr, M.L.A., rose to the first presidency with a profound knowledge of biochemistry from the ranks of the general practitioners, and others have achieved an international reputation, especially Dr. Michael Kelly. The opportunities are here in Australia to do some original research in rheumatology to stimulate the backbone of our profession, the general practitioners, in the early recognition of rheumatic disease before the stage of irreversibility and tragic deformities. Where is the hold-up? Is it petty jealousy, as stated by the late Sir Trent de Crespigny after his visit to research centres in the U.S.A.? Those of us who have had the privilege to see the amazing drive and organization behind the American Rheumatism Association and that of similar bodies in England and on the Continent during the past ten years would agree with Dr. Michael Kelly to see the results of suitable hormone injections to resuscitate the Australian Rheumatism Council from its state of dormancy before approaching death.

Yours, etc.,

175 Brougham Place,  
North Adelaide,  
South Australia.  
January 1, 1959.

PHILIP ALPERS.

#### MEDICINE IN CHINA.

SIR: In answer to the question raised in "Current Comment" on "Medicine in China" (M.M. J. AUST., November 15, 1958), Dr. McClelland in your issue of December 6 cites Chinese chauvinism as the cause of the keen interest now being taken by the Chinese Government in traditional medicine and its practitioners. But, while this is an indubitable and very obtrusive factor, it is easily understandable by anyone familiar with China's century and a half of contacts with the West; and a much more potent cause is dire necessity.

As I have before described,<sup>1</sup> the medical missionary body which laid the foundation of modern medicine in China worked for a century to establish a high professional standard of Christian medical practice in that country, and to train, in their six medical schools of this half-century, a nucleus of Chinese doctors who would carry on the tradition that they established. And they did just that.

But the problem of the present Government's Department of Health is quite different. How can it most effectively and quickly raise the deplorable standard of health of its whole population of 600,000,000 people? The total number of modern doctors available from every source would probably be nearer 5000 than 10,000. But the number of practitioners of traditional medicine, dispersed widely throughout the whole country, is about half a million. It is surely obvious that, unless the Government can win the confidence and cooperation of this latter body, its already formidable task will be many times more difficult.

The Chinese attitude under such circumstances differs from the Western. In the late thirties there were in China only some few hundred Western trained obstetric nurses; but they were very highly trained, as they had to be prepared to treat, far from all medical aid, the worst emergencies, often frightfully torn and infected by the ministrations of the village "Sally Gamps", of whom there were naturally many scores of thousands. So the Nationalist Government of those days began to call in the latter by groups for refresher courses of a few weeks, during which obstetric nurses taught them the barest rudiments of common cleanliness, asepsis, puerperal management and infant care.

The Government of the People's Republic, this time with a medical rather than a nursing problem, is showing a precisely similar behaviour pattern. And this is paying very high dividends. Already 20,000 of the traditional practitioners have offered for training and entered the Government Health Service. And during the six or seven years that the latter has been in full operation, the reduction in

infectious diseases—cholera, ancylostomiasis, the dysenteries, the typhoids, schistosomiasis, infantile summer diarrhoea, etc.—has been spectacular.

But it would show a complete misunderstanding of the situation to infer that the Government is neglecting modern medicine. Last year my brother visited Central China and met a number of my former colleagues at the Union Hospital, Hankow. Its accommodation has now grown from 215 to 1000 beds, and it has become the practising hospital for the new Central China Medical School with about 1000 students. A number of our old staff are now in high positions of responsibility in the new hospital and school, including that of medical superintendent and deputy medical superintendent. This set-up is typical of the Government's attitude in the various new medical schools it is establishing and in the public health service it is building up. There is no question of religious discrimination. All members of our old Western trained staff—doctors, nurses, technicians, etc.—are in urgent demand, comparatively well paid and mostly in positions of responsibility. But it should be obvious that, with all the will in the world, it must be long before such a bottle-neck of modern-trained personnel can be overcome and sufficient training schools established.

Not all Chinese leading doctors, however, are employed in teaching and public health work. A recent number of the *Chinese Medical Journal*<sup>2</sup> carried an account of the work of a new heart clinic in Shanghai. There was an interim report on the progress of the first several hundred mitral commissurotomies that had been done to date, and the results were roughly comparable with those in similar reports on cardiac surgery in the West.

As to ancient Chinese medicine—at the biennial conference of the China Medical Association held in Hong Kong in the late thirties, a memorable paper was read by Bernard Read, the pharmacologist of the Peking Union Medical College. He had in the previous six months worked through a medicinal plant, *Ephedra vulgaris* (*ma huang* of the Chinese pharmacopoeia), which for two thousand years had been very famous in Chinese medicine, and he had isolated and tested out ephedrine, the first active principle of a plant to replace a living hormone.

There recently returned to Australia a young graduate from one of our medical schools who had spent the previous two years under treatment by acupuncture in Peking for rheumatoid arthritis of both hands and fingers. There were still huge enlargements of most of the joints (I do not remember to have seen larger); but flexibility and movement were nearly perfect, there was no trace of fibrous thickening, and when I saw him he was refreshing his surgical experience with a view to beginning in private practice.

I have seen horrible things done by inexperienced acupuncture; but in medicine it does not do to be too didactic.

Yours, etc.,

Bathurst,  
N.S.W.,

H. OWEN CHAPMAN.

December 24, 1958.

#### HYPNOSIS: AN EVALUATION OF ITS PLACE IN MEDICINE.

SIR: Dr. Ainslie Meares, in his provocative lecture on hypnosis in medicine (M.M. J. AUST., December 27, 1958), emphasizes its dangers. There are, of course, dangers in it, as there are in other forms of therapy, such as the aspirating of a chest, inducing convulsions, injecting penicillin or performing an operation. I submit that the danger of hypnosis is being over-stressed, especially as "light" hypnosis suffices for most purposes; indeed, most people never pass beyond this light stage. As Dr. Meares says, it is in deep hypnosis that trouble is most likely to occur. Dr. Meares discusses the specific dangers—namely, perverse motivation, unfulfilled post-hypnotic suggestion and traumatic insight. Are not these due, not to hypnosis itself, but to errors of technique or judgement on the part of the hypnotist? It is the surgeon, not surgery, who is responsible for a slipped ligature or a faulty anastomosis. Dr. Meares speaks of people seeking hypnosis for perverse reasons. They may also seek some other forms of psychotherapy or even surgery for some perverse reason, and it is the doctor's difficult task to uncover such people and help them by the best means. Next, the hypnotist's perverse motivation is considered. But

<sup>1</sup> M.M. J. AUST., 1948, 2: 10 (July 3).

<sup>2</sup> Chin. med. J., 1957, 75: 899 (October).



not only the hypnotist must watch himself here, but every one of us. "It would seem clear", says Dr. Meares, "that no physician should hypnotize a patient without full consideration of his own and the patient's motivation." I think that the word "hypnotize" should be replaced by the word "treat" used in the widest sense.

Unfilled post-hypnotic suggestions are, as Dr. Meares's examples show, suggestions that should never have been given; the hypnotist, not hypnosis, is to blame.

Traumatic insight is the third danger mentioned by Dr. Meares. Not many medical hypnotists seem to have encountered it. I conclude with an extract from a personal letter from Professor T. Ferguson Rodger, who was chairman of the subcommittee that issued a report for the British Medical Association in 1955 on the "Medical Use of Hypnotism" (Appendix X in the Supplement of the *British Medical Journal*, April 23, 1955):

I remember having a conversation with Dr. Meares at Zurich last year, and following it I asked the B.M.A. to make available to him Press reports on the ill effects of hypnosis conducted by stage hypnotists and the like. I cannot recall, however, any cases having been reported where ill effects had followed its use by medical men. This would suggest that, provided hypnosis is properly undertaken, with due precautions, there can be no objection to its use by surgeons and other medical men.

As a result of this report stage demonstrations of hypnosis became illegal in Great Britain, and I respectfully suggest to Dr. Meares that, without discouraging further research of its use in medicine, he use his prestige and influence to have similar performances prohibited in this country.

Yours, etc.,

185 Macquarie Street,  
Sydney,  
December 30, 1958.

M. P. SUSMAN.

#### THE PATIENT, THE SURGEON AND THE ANAESTHETIST.

SIR: In reply to M. P. Susman's question (*MED. J. AUST.*, January 3, 1959), the anaesthetist's response should be: "Yes, unless local anaesthetic would be safer." Where general practitioners are concerned, a choice of role may be offered: "We will have to operate; which one of us will anaesthetize?" A refinement is to toss for the head of the table.

Yours, etc.,

398 Crown Street,  
Wollongong,  
N.S.W.  
December 29, 1958.

M. C. MCKINNON.

SIR: An anaesthetist, like most doctors in clinical practice, is responsible to his patients, possibly their next-of-kin and the Crown, for his own professional actions. His response to Dr. Susman's question (*MED. J. AUST.*, January 3, 1959) should be: (i) When he concurs in the surgeon's professional judgement: "Yes". (ii) When he has insufficient special knowledge to judge personally the merits of the case, but enough confidence in the surgeon's ability to enable him to accept the surgeon's decision, endorse it and make it his own: "Yes". (iii) When these conditions do not apply: "No".

Yours, etc.,

Canberra, A.C.T.  
January 3, 1959.

DOUGLAS VANN.

#### THE COMPENSATION OF INDUSTRIAL INJURIES.

SIR: Dr. Macdonald Tow's system (*MED. J. AUST.*, December 6, 1958, page 767) appears temptingly simple, and it is sufficiently vague and ill-defined to allow almost everyone who has not recovered from a compensable injury to qualify as mentally weak or subconsciously dishonest.

But what of injuries which are not compensable? And what of skeletal pains which have not followed injuries? Intractable backache, for instance, is more common in

women than in men, and the clinical picture is the same in those who have not been injured as in those who have.

This is not a new problem. It is as old as medicine; and in the past, as at present, it has been interpreted in the light of shifting fashion. Strangely, too, those fashions have always permitted the doctor to blame the patient for not getting well.

The present fashion of treating these patients as psychiatric or as malingerers is disastrous. Extreme unhappiness has been caused in at least one family by actions of a husband who became unsympathetic after his wife's "neurotic" backache had been "explained" by a psychiatrist. The pain is hard enough to bear without the added bitterness of knowing oneself disbelieved.

Yours, etc.,

M. KELLY.

410 Albert Street,  
East Melbourne,  
Victoria.  
December 22, 1958.

#### ADVERTISING MATTER AND THE MAILS.

SIR: The dangerous abandon with which drugs are advertised on envelopes and postcards or sent unasked for can best be overcome, we suggest, by the doctors concerned marking the items in red ink: "Return to sender. Addressee cannot conscientiously accept this imprudent mailing."

Lest we be justly called hypocrites, let us first check (i) that we cover the offending information before posting, and (ii) that we give post office box numbers, or lock our letter boxes when ordering drugs, or have parcels too big for the letter box sent by registered post.

Yours, etc.,

D. EVERINGHAM.

The Talbot Clinic,  
21 East Street,  
Rockhampton,  
Queensland.  
December 23, 1958.

#### THE JACKSON LECTURE—PROGRESS IN PSYCHIATRY.

SIR: Having recently read Ernest Jones' on the life and works of Sigmund Freud, I find Dr. G. B. Murphy's reference in the recent Jackson Lecture (*MED. J. AUST.*, January 3, 1959) curious in its emphases. He calls Freud "an intelligent imaginative neurotic Jew" and quotes fainting spells as a symptom.

Apart from the inevitable emotional reaction that such a statement must have produced in his audience (surely Dr. Murphy would have realized this!), in fact there is little overt evidence of neurosis in Freud's life. The fainting spells from which he "suffered" occurred only on two occasions, provoked by emotional stresses.

If you look for neurosis in anyone you will find it, and psychiatrists are no exception. An honest one will admit readily that this disorder is actually responsible for his choice of occupation. In Freud's life, on the contrary, there is much that would have rendered the average "healthy" man distinctly and overtly neurotic. Jones shows clearly that Freud's last 16 years, with their ordeal of 33 successive operations on a constantly painful and obviously malignant growth, would have been borne by few of us with a courage and acceptance equal to Freud's.

Freud's social insight comes under Dr. Murphy's fire because he "had faith in the U-boat war and in the ultimate victory of Germany in World War I". Surely this faith was highly justified early in the war! By March, 1918, however, he wrote to a colleague:

I suppose we have to wish for a German victory and that is (1) a displeasing idea and (2) still improbable.

Dr. Murphy stresses Freud's "enthusiastic" presentation of his material, as if this were a fault—but is it not characteristic of true creativeness in any sphere?

<sup>1</sup> "The Life and Works of Sigmund Freud", Volume II, page 220.

All in all, I cannot help finding his words generally belittling, his admiration very grudging and his whole attitude rather mean. A poet,<sup>1</sup> less chary in his gratitude, wrote that, when Freud died,

Only Hate was happy, hoping to augment  
His practice now, and his shabby clientèle  
Who think they can be cured by killing  
And covering the gardens with ashes.  
They are still alive but in a world he changed  
Simply by looking back with no false regrets;  
All that he did was to remember  
Like the old and be honest like children.

Yours, etc.,

A. M. LIEBHOLD.

65 Chapel Street,  
St. Kilda,  
Victoria.  
January 3, 1959.

## Obituary.

GEORGE ATKINSON.

We are indebted to SIR WILLIAM UPJOHN for the following account of the career of the late Dr. George Atkinson.

George Atkinson, who died on August 14, 1958, was well known in many parts of Victoria. He was born in 1885, and later became a scholar at Xavier College, Kew, where he was very well liked by his fellow scholars for his cheerful, sunny nature, and for his prowess at games, especially football and cricket. He maintained his interest and skill in football, and in his youth he played League football with Collingwood. Later in life he had to give up football and cricket, but games always interested him, and whenever he could find time, he played tennis or golf.

In 1910 he qualified M.B., B.S. at the University of Melbourne, and later served on the resident staff of St. Vincent's Hospital, and at the Women's Hospital. He was senior resident medical officer and superintendent at the Women's Hospital in 1912. For several years he attended as a physician in the out-patients' department at St. Vincent's Hospital, Melbourne.

During the 1914-1918 war he joined the Australian Army Medical Corps, and was for a while the medical officer at Langwarren camp. At that time there was an epidemic of meningitis in the military camp, and he himself caught the infection. After a hazardous period he made a good recovery. He was discharged to civil life, and then commenced practice in the country.

Few practitioners can have had first-hand experience of private practice in so many parts of Victoria as he did. He practised for varying periods in Murchison, Meredith, McArthur, Hamilton, Learmonth, Murrumbidgee, Brighton and St. Kilda. In all of these places he made many friends. When war broke out in 1939 he was practising in Collins Street, Melbourne.

In 1940 he joined the staff of the Australian Mutual Provident Society as its city medical referee, which position he held till 1954.

George Atkinson always appeared younger than his years, and maintained his athletic appearance, alert and cheerful expression and friendly manner till well advanced in years. Most of his medical student contemporaries are dead; but he is remembered with affection by those who survive, and by the many friends he made in the numerous localities where he had practised his profession.

DR. W. W. S. JOHNSTON writes: The recent death of Dr. George Atkinson has removed one who had endeared himself to many colleagues and friends by his kindly nature and his eager participation in the numerous activities associated with his lengthy and energetic life. As a medical student his interests were varied; he was a prosector in 1908 and a member of committee of the Medical Students' Society in 1909-1910. He was a keen footballer, particularly in the hotly contested inter-faculty matches for the Baldwin Spencer Cup.

After qualifying, he was attached to both St. Vincent's Hospital and the Women's Hospital in various capacities. He acted as medical superintendent at the latter institution in 1912, and it was here that his interest in gynaecology was roused. However, any plans he may have formed to specialize

in that field were nullified by the outbreak of the first World War. He enlisted for active service, and while in camp contracted cerebro-spinal meningitis. After a severe and prolonged illness he recovered, but this event set the pattern for his future life. He was unfit for further military service, and was advised by Sir Richard Stawell to commence general practice in a quiet country district. This he did at Learmonth, near Ballarat. With an aptitude for attracting patients and a deservedly high reputation in his professional work, he rapidly built up a large practice. Later he went to Ballarat, and from there moved in succession to a number of different areas, in each displaying a flair for achieving success and gaining the confidence of his patients. His last and longest period of practice was that spent with the Australian Mutual Provident Society. For many years, commencing in 1940 and continuing till the end of 1954, he held the full-time appointment of city medical referee at the Society's Melbourne branch office, and during that time examined thousands of applicants for life assurance. The speed and accuracy with which he worked, missing no relevant points in history or physical examination, were remarkable, and made him the ideal person for such a position. He also acted in a medical advisory capacity to members of the staff, and as such his friendly and sympathetic outlook made him their firm friend as well as valued doctor.



A tribute should be paid to his devotion to his mother. His father had died when George Atkinson was a young man, and from then the son took over the responsibility of ensuring that his mother lacked nothing that he could provide for her well-being. Every action was planned with her interest in view, and this completely selfless attitude continued unabated until her death some few years ago. In 1952 he married Mrs. Marjorie Prudhoe, and it seemed that a lengthy period of happy and well-earned rest lay ahead. But ill-health overtook him, and for the last twelve months he was an invalid. To his widow, his friends offer their deep sympathy in the loss of one who was a loyal comrade, a faithful worker and a man of courage in adversity.

THE REVEREND FATHER J. R. BOYLEN, S.J., Rector of Xavier College, writes: Dr. George Atkinson was a boy at Xavier College during the years 1903 and 1904. An old friend writes of him: "A very loving and lovable fellow who put into act Tennyson's lines:

"Because right is right, to follow right  
Were wisdom in the scorn of consequence."

A prominent Old Xavierian, asked for the opinion he held of him 54 years after their fellowship at Xavier, said: "George Atkinson was one of Nature's gentlemen, one always to be looked up to." Referring to his ability at games, the sports master of the day has left on record two appreciations:

In Football, George Atkinson was a fine player, a splendid mark and kick, clever at dodging, and fast with the ball. Not the least pleasing feature of his play was his absolute fairness.

In Cricket, he was a fair all-rounder in batting, bowling and fielding, and these would have been much improved had not an accident on the football field kept

<sup>1</sup> Auden, W. H., "In Memory of Sigmund Freud".



him out of action during the closing term of his last year at school.

After leaving school at Xavier, Atkinson went to the University of Melbourne to study medicine. In 1909 he took his degree. For some time he was on the medical staff at the Women's Hospital, and later did private practice at Skipton and Warrnambool. His death on August 14, 1958, left the medical world and the hearts of those who knew him poorer places. May he rest in peace.

## Post-Graduate Work.

### THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

#### PROGRAMME FOR 1959.

THE Post-Graduate Committee in Medicine in the University of Sydney announces the following programme of post-graduate activities for 1959.

#### Metropolitan and Week-End Courses.

The following metropolitan and week-end courses will be held: February 14 and 15, "Art of Teaching" (limited); February 25 to March 3, anaesthetics (St. Vincent's Hospital), by Professor W. W. Mushin, Cardiff (see separate announcement); March 14 and 15, neurology; March 21 and 22, electrocardiography; April 10, 11 and 12, respiratory diseases (guest lecturer, Sir Geoffrey Todd); April 13 to 17, "Recent Advances in Mental Deficiency and the Care of Physically Handicapped Children" (Newcastle), by Dr. L. T. Hilliard, London (see separate announcement); May 11 to 22, general revision; June 6 and 7, rheumatic diseases; June 20 and 21, renal diseases; June 27 and 28, Rachel Forster Hospital (medical); July 4 and 5, gastro-intestinal diseases; July 11 and 12, "Specials"; July 25 and 26, radio-isotopes; August 1 and 2, paediatrics; August 8 and 9, psychosomatic medicine; August, "Reunion Week", Royal North Shore Hospital; August 31 to September 4, gynaecology and obstetrics; September 7 to 18, "Reunion Week", Royal Prince Alfred Hospital; September 21 to 25, Old Sydney Hospitalers' Week; September 26 and 27, mental deficiency; September 29 to October 3, "Reunion Week", St. Vincent's Hospital; October 10 and 11, Rachel Forster Hospital (surgical); November, anaesthetics, Sydney Hospital (limited).

Consideration is being given to holding courses in "Civil Defence Aspects of Nuclear Medicine", "Ear, Nose and Throat Cancer" (to coincide with the visit of Dr. John J. Conley), "Traumatic Surgery" and "Occupational Medicine".

#### Annual Subscription Course.

A course in anaesthetics is being arranged around the visit of Professor William W. Mushin, Professor of Anaesthetics, Welsh National School of Medicine, at St. Vincent's Hospital, from Wednesday, February 25, to Tuesday, March 3. At 8.30 each morning, Professor Mushin will conduct a practical demonstration, attendance at which is limited, and at 2 o'clock each afternoon he will hold informal discussions at which attendance is unlimited, all medical practitioners being invited to be present. The course is open to members of the annual subscription course without charge. Those wishing to attend are requested to advise the Committee not later than February 6, 1959.

A course of three lectures entitled "The Hospital and the Community" will be held at the Mater Misericordiae Hospital, North Sydney, at 8.15 p.m. on the following Wednesdays: March 4, "The Changing Pattern of Hospital Services"; March 11, "Socio-Economic Aspects of Hospital Services"; March 18, "The Hospital as a Factor in Medical Practice".

A course in "Recent Advances in Mental Deficiency and the Care of Physically Handicapped Children" will be held in Newcastle from April 13 to 17, to coincide with the visit of Dr. L. T. Hilliard, Consultant Psychiatrist and Physician Superintendent at the Fountain Hospital, London. The course will be limited to 20 selected candidates, and will include visits to various mental institutions in the area.

The fee for attendance at the annual subscription course, which covers attendance at lectures by overseas visitors and other specially arranged activities, is £2 2s., or £1 1s. for first and second year resident medical officers, from July 1. A detailed diary card is printed at regular intervals throughout the year and posted to members. Occasionally last-

minute alterations to meetings are necessary, and these are notified by advertisement in *The Sydney Morning Herald* ("Public Notices"), if possible on the day before the meeting.

#### Resident and External Training Facilities.

The following facilities are available:

**Anaesthetics.**—Post-graduate residencies are available for practical training in modern methods of anaesthetics at The Royal Newcastle Hospital. Fees, including board and residence, are £5 15s. 6d. per week of five days, Monday to Friday. No vacancies are available before 1960. Post-graduate residencies for full-time residential training in anaesthetics are also available at the Lewisham Hospital for a minimum period of three weeks during the months of June, September and December, 1959. Fees, including board and residence, are £6 6s. per week. A full-time course in anaesthetics, non-resident, will be conducted by the Department of Anaesthetics and Resuscitation, Sydney Hospital, in November, 1959.

**Blood Grouping and Transfusion.**—Instruction is arranged, free of charge, at the Red Cross Blood Transfusion Service, or The Royal Newcastle Hospital.

**Gynaecology and Obstetrics.**—Post-graduate residencies in gynaecology and obstetrics are available at The Women's Hospital, Crown Street, and at the Royal Hospital for Women, Paddington. Fees, including board and residence, are £5 15s. 6d. per week of seven days.

**External Studies.**—Arrangements can be made with due notice to meet the individual needs of medical practitioners requiring *à la carte* courses. Fees are £1 1s. enrolment and £1 1s. per week.

**General Residencies for Women Graduates.**—These are available at the Rachel Forster Hospital for Women and Children, Redfern. The fee is £6 6s. per week of seven days.

**Post-Mortem Examinations.**—Instruction in the technique of the performance of post-mortem examinations can be arranged free of charge.

#### Week-End Conferences, Country Centres.

The following dates and centres are all subject to confirmation: April 18 and 19, Bathurst; April 18 and 19, Newcastle (gynaecology and obstetrics); June 6 and 7, Tamworth; June 13 and 14, Broken Hill; June 27 and 28, Katoomba; July 4 and 5, Hornsby; August 15 and 16, Newcastle (medicine); September 12 and 13, Taree; October 10 and 11, Wagga Wagga; October 17 and 18, Albury; October 17 and 18, Farramatta; October 24 and 25, Newcastle (surgery); November 14 and 15, Bega-Cooma.

#### Diploma Courses.

The following diploma courses will be held: January 12, course in advanced medicine (11 weeks); March 2, course in advanced surgery (8 weeks); March 16, D.D.R. I (4 months), D.T.R. I (4 months); May 11, D.D.M. I (12 weeks); June 2, advanced medicine (11 weeks); June 8, D.P.M. I (3 months); July 6, D.A. II (3 months), D.C.P. Group I (7 months); July 20, D.P.M. II (7 months), D.D.R. II (8 months), D.T.R. II (8 months); August 10, D.D.M. II (7 months); August 31, D.A. I (11 weeks), D.G.O. I (11 weeks), D.L.O. I (11 weeks), D.O. I (11 weeks); September 21, D.C.P. Group II (6 months), D.C.P. Group III (6 months); November 16, D.G.O. II (3 months), D.L.O. II (3 months), D.O. II (3 months).

#### Histological Slides.

A limited number of sets of slides of normal histology suitable for candidates working for degrees and diplomas are now available at £4 4s. a box. Each box contains about 75 slides. Applicants should indicate the examination for which they are studying.

#### General Information.

Dates of all courses should be confirmed with the Course Secretary, The Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney (telephone BU 4497-98), from whom further details regarding fees, diploma regulations and other information may be obtained. Telegraphic address: "Postgrad Sydney."

#### Annual Subscription Course.

The Post-Graduate Committee in Medicine in the University of Sydney announces that Professor W. W.

<sup>1</sup> Arranged by the N.S.W. State Committee, Royal Australasian College of Surgeons, in conjunction with the Department of Surgery.

Mushin, M.A., F.F.A.R.C.S., D.A., Professor of Anaesthetics, Welsh National School of Medicine, Royal Infirmary, Cardiff, Wales, will conduct a course in anaesthetics at the St. Vincent's Hospital from Wednesday, February 25, to Tuesday, March 3, 1959. At 8.30 each morning Professor Mushin will conduct a practical demonstration, and at 2 o'clock each afternoon he will hold informal discussions. The morning demonstrations will be strictly limited, and are open to members of the annual subscription course without charge. Those wishing to attend are requested to advise the Committee not later than February 6, 1959. Attendance at afternoon discussions is unlimited, and all medical practitioners are invited to be present.

The course will conclude with a lecture by Professor Mushin entitled "Anaesthesia and the Bad Risk Patient" on Tuesday, March 3, at 8.15 p.m., in the Stawell Hall, 145 Macquarie Street, Sydney. This lecture is being held in conjunction with the Australian Society of Anaesthetists (N.S.W. Section) and the Faculty of Anaesthetists, Royal Australasian College of Surgeons (N.S.W. State Committee).

Inquiries should be directed to the Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. Telephones: BU 4497-8.

#### Week-End Courses for General Practitioners.

The Post-Graduate Committee in Medicine in the University of Sydney announces the following week-end courses for general practitioners, to be held in Sydney during March, 1959: Neurology, on Saturday, March 14, from 2 to 5 p.m., and Sunday, March 15, from 9.45 a.m. to 5.30 p.m., at The Royal North Shore Hospital, of Sydney; electrocardiography, on Saturday, March 21, from 10 a.m. to 5 p.m., and on Sunday, March 22, from 10 a.m. to 1 p.m., at Sydney Hospital.

The fees for attendance at each of these courses are £3 3s., and detailed programmes will be published shortly. Early application, enclosing remittance, should be made to the Course Secretary, Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. Telephones: BU 4497-4498. Telegraphic address: "Postgrad Sydney."

#### THE MELBOURNE MEDICAL POST-GRADUATE COMMITTEE.

##### PROGRAMME FOR FEBRUARY, 1959.

#### Gynaecology and Obstetrics Refresher Course for Recent Graduates.

A REFRESHER COURSE in gynaecology for recent graduates will be conducted from February 9 to 20, 1959, at the Royal Women's Hospital, Swanston Street, Carlton. This will be a full-time course, consisting of lectures, demonstrations and ward rounds by members of the hospital staff. It will be desirable for those taking part to wear long white coats while in the wards. The fee is £10 10s. for the whole course, payable to the Committee with enrolment on their special form, which can be obtained on request. Commencement of the course depends on receipt of a satisfactory number of enrolments by January 22.

#### Overseas Lecturers.

On Thursday, February 12, at 8.15 p.m., in the Medical Society Hall, Professor Alan Johnstone, of the University of Leeds, will give an open lecture to the medical profession on "Milestones in Radiology"—a lecture which will be of interest to general practitioners. Attendance is by the payment of an annual subscription to the Committee, or a fee of 15s.

On Monday, February 23, at 8.15 p.m., in the Medical Society Hall, Mr. W. Gissane, F.R.C.S., of Birmingham, will give an open lecture to the medical profession on "Treatment and Prognosis in Burns, with some Observations on the Prevention of 'Home Accidents'". Mr. Gissane, who will be in Melbourne from February 18 to March 4, is the Sir Arthur Sims Commonwealth Travelling Professor for 1959. Details of his visit are in the hands of the Royal Australasian College of Surgeons. The above-mentioned lecture is without fee.

#### Anatomy.

On February 16, at 2.15 p.m., classes will begin in anatomy, suitable for candidates for Part I of M.S., F.R.A.C.S., F.F.A.R.C.S., D.A., M.G.O., D.O., D.L.O., D.D.R., D.T.R., D.C.R.A. and D.P.M. These will be held at the University

of Melbourne on Mondays and Wednesdays, till August. (The time may vary for neuroanatomy to suit the class.) The fee for each course is £42, and enrolments on the Committee's form should be received by them before February 2.

#### Physiology and Pathology.

Courses in physiology and pathology will commence on March 2, and enrolments are due by February 16. They also will be held on Monday and Wednesday afternoons.

#### Country Courses.

**Warragul.**—On February 14, at the Base Hospital, Warragul, the following course will be held: 2.30 p.m., "Varicose Veins", Mr. F. W. Connaughton; 4 p.m., "Surgery of the Anus and Rectum", Mr. E. S. R. Hughes; 8 p.m., quiz session, followed by supper at the hospital. Dr. P. M. Dow, 57 Victoria Street, Warragul, telephone 3, is the local secretary. He will arrange any accommodation required.

**Bendigo.**—On February 27, at Lister House, 37 Rowan Street, Bendigo, at 8 p.m., Mr. J. Woodward will lecture on "Management of Common Fractures of Limbs". The local secretary is Dr. M. Clark, 98 Mitchell Street, Bendigo.

#### Flinders Naval Depot.

On February 18, at Flinders Naval Depot, at 2.30 p.m., a lecture will be given by Dr. Bruce Robinson by arrangement with the Royal Australian Navy.

#### RECORDED LECTURES.

The following are additions to the Melbourne Medical Post-Graduate Committee's library of recorded lectures, catalogued in their syllabus in December: "The Responsibility of the Family Doctor in the Medical Care of Old Persons", by Dr. Marjory Warren, of London (two disks, 21 slides); "The Aims of an Industrial Health Service", by Dr. A. Austin Eagger, of Slough, England (one disk only). These may be obtained from the Committee on request.

#### INFORMATION.

The address of the Melbourne Medical Post-Graduate Committee is 394 Albert Street, East Melbourne, C.2. Telephone: FB 2547.

## Congresses.

#### FIFTEENTH INTERNATIONAL TUBERCULOSIS CONFERENCE.

The fifteenth International Tuberculosis Conference will be held from September 11 to 18, 1959, in Istanbul, Turkey. The chairman will be Professor Tevfik Caglam, of Turkey. Medical practitioners who will be in Europe during that period, and who would be interested to attend the conference, are invited to send their names and plans to the Executive Secretary, N.A.P.T.A., 188 North Terrace, Adelaide, so that the Council may be able to assist them as far as possible.

The complete programme of the conference, together with registration forms, may be obtained on application to the Secretariat of the International Union against Tuberculosis, 15 Rue Pomeru, Paris, XVI\*, France.

#### WORLD CONFEDERATION FOR PHYSICAL THERAPY.

##### Third World Congress.

The third World Congress of the World Confederation for Physical Therapy, of which the Australian Physiotherapy Association is a member organization, will be held in Paris in September, 1959. The theme will be "Physical Therapy: Its Importance in Human, Economic and Social Development". The principal subjects to be included in the scientific programme will be as follows: neurology, geriatrics, orthopaedic surgery and traumatology, rheumatology, heart and chest conditions, aids and prostheses, psychiatry. The scientific sessions will be held in the new Faculty of Medicine, 45 Rue des Saints-Pères, Paris, VI\*, and there will be simultaneous translation of the lectures and discussions in English, German and French.



Any medical practitioner who is willing to lecture is asked to get into touch with the Federal Council of the Australian Physiotherapy Association, 83 William Street, Melbourne, C.I., Victoria, supplying name and full particulars, together with full title of the subject chosen.

## Royal Australasian College of Surgeons.

### GORDON CRAIG FELLOWS: GRANT-IN-AID.

A small grant-in-aid for an approved research project may be awarded by the Council of the Royal Australasian College of Surgeons at its meeting in February, 1959. Applicants are asked to communicate with the Secretary of the College at Spring Street, Melbourne, C.I., Victoria, and provide him with full details of the nature of the work which they are undertaking, or propose to undertake, together with information concerning the availability of other funds, etc.

## Notes and News.

### Prize for a Medico-Surgical Film.

The annual prize awarded by *La Presse Médicale* for a medico-surgical film, amounting to 100,000 francs (with the possibility of its being divided), and various other awards, will be presented during the last session of the course on *Actualités médico-chirurgicales* at the new Faculty of Medicine, Paris, on March 17, 1959. The judges will consider the instructional value of the film, as well as its purely cinematographic quality. Only film of 16 millimetre size will be accepted. Entries and films should be sent before February 15, 1959, to *Secrétariat du journal La Presse Médicale*, 120 Boulevard Saint-Germain, Paris VI\*. The authors of the best films will receive awards. All films are eligible for awards, including those subsidized or produced by a laboratory or firm.

### Department of Health, Victoria.

The Department of Health, Victoria, has opened a branch office at 19 Park Street, South Yarra (telephone BM 1201), and the following services will operate from that address from December 15, 1958: B.C.G. Unit; distribution of immunizing material, including Salk vaccine; Poliomyelitis Division; School Medical Services.

### Clinico-Pathological Conferences at Sydney Hospital.

Clinico-pathological conferences will be held as usual at Sydney Hospital during 1959. These conferences are held in the Maitland Lecture Theatre on the third Tuesday of each month at 5 p.m. The first conference will be held on January 20.

## Naval, Military and Air Force.

### APPOINTMENTS.

The following appointments, changes, etc., are published in the *Commonwealth of Australia Gazette*, No. 78, of December 23, 1958.

### NAVAL FORCES OF THE COMMONWEALTH.

#### Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

##### Emergency List.

*Transfer to the Retired List.*—Surgeon Captain James Martin Flattery, O.B.E., is transferred to the Retired List, dated 2nd November, 1958.

#### Citizen Naval Forces of the Commonwealth.

##### Royal Australian Naval Volunteer Reserve.

*Termination of Appointment.*—The appointment of the following is terminated to date 31st March, 1958: Surgeon Lieutenant-Commander Donald Kerr Grant.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED DECEMBER 13, 1958.<sup>1</sup>

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism ..	1	2(2)	4(2)	..	..	..	..	..	7
Amoebiasis ..	..	..	..	..	..	..	..	..	..
Ancylostomiasis ..	..	..	..	..	..	..	6	..	9
Anthrax ..	..	..	..	..	..	..	..	..	..
Bilharziasis ..	..	..	..	..	..	..	..	..	..
Brucellosis ..	1	..	1	..	..	..	..	..	2
Cholera ..	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus) ..	..	..	..	..	..	..	..	..	..
Dengue ..	..	..	..	..	..	..	..	..	..
Diarrhoea (Infantile) ..	3(1)	11(11)	4(4)	..	1	..	4	1	24
Diphtheria ..	..	..	..	..	..	..	7	..	12
Dysentery (Bacillary) ..	..	5(5)	..	..	..	..	..	..	1
Encephalitis ..	1	..	..	..	..	..	..	..	..
Filariais ..	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice ..	..	..	..	..	..	..	..	..	..
Hydatid ..	..	..	..	..	..	..	..	..	..
Infective Hepatitis ..	94(39)	33(16)	9(3)	35(7)	1(1)	..	2	..	174
Lead Poisoning ..	..	..	..	..	..	..	..	..	..
Leprosy ..	..	..	..	..	..	..	..	..	..
Leptospirosis ..	..	..	1	..	..	..	..	..	1
Malaria ..	..	..	..	..	..	..	1	..	1
Meningococcal Infection ..	3(3)	1	..	..	..	..	..	..	4
Ophthalmia ..	..	..	..	..	..	..	..	..	..
Ornithosis ..	..	..	..	1(1)	..	..	..	..	1
Paratyphoid ..	..	..	..	1	..	..	..	..	1
Plague ..	..	..	..	..	..	..	..	..	..
Poliomyelitis ..	..	3(1)	..	..	..	..	..	..	3
Pyrexial Fever ..	1	11(1)	..	..	..	..	..	..	2
Eubella ..	..	43(38)	1(1)	3(3)	62(61)	..	..	..	119
Salmonella Infection ..	..	..	..	2(2)	1(1)	..	1	..	4
Scarlet Fever ..	9(5)	14(10)	2(1)	3(3)	1	..	..	..	29
Smallpox ..	..	..	..	..	..	..	..	..	..
Tetanus ..	..	..	..	..	..	..	..	..	..
Trachoma ..	..	..	..	..	1(1)	..	5	..	6
Trichinosis ..	..	..	..	..	..	..	..	..	..
Tuberculosis ..	35(23)	13(15)	11(6)	9(3)	7(5)	5(1)	1	..	84
Typhoid Fever ..	1(1)	..	..	..	..	..	..	..	1
Typhus (Glas. and Tick-borne) ..	..	..	1	..	..	..	..	..	1
Typhus (Louse-borne) ..	..	..	..	..	..	..	..	..	..
Yellow Fever ..	..	..	..	..	..	..	..	..	..

<sup>1</sup> Figures in parentheses are those for the metropolitan area.

## Honours.

### NEW YEAR HONOURS.

The following medical practitioners have been included by Her Majesty the Queen in the New Year Honours List;

Dr. Arthur William Morrow, of Sydney, has been created a Knight Bachelor.

Dr. John Alexander James, of Canberra, and Dr. Alan Bruce Lilley, of Sydney, have been created Commanders of the Most Excellent Order of the British Empire.

Dr. Janet Pierson Cooper and Dr. Cyril Joseph Tonkin, of Melbourne, and Dr. Peter Roylance Delamothe, of Bowen, Queensland, have been created Officers of the Most Excellent Order of the British Empire.

Dr. Joseph Andrew Arratta, of Muttaborra, Queensland, has been created a Member of the Most Excellent Order of the British Empire.

## Notice.

### THE POST-GRADUATE MEDICAL FOUNDATION OF THE UNIVERSITY OF SYDNEY.

The following is a list of donations so far received by the Post-Graduate Medical Foundation of the University of Sydney.

Mr. F. Klement, £100; Mr. E. R. Williams, £500; Pfizer Pty. Ltd., £250; Australian Consolidated Press Ltd., £3000; Watt Street Hospital Handicapped Children's Welfare Association, £500; Parke, Davis and Co. Ltd., £1500; Johnson and Johnson Ltd., £250; "Truth and Sportsman" Ltd., £200; Australian Consolidated Press Ltd., £2000; Dr. Austin Callen, £5 5s.; Medical Representatives Organization, £31 10s.; Pfizer Pty. Ltd., £1500; Schering Corporation, U.S.A., £100.

## Nominations and Elections.

The undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Bookallil, Michael John, M.B., B.S., 1958 (Univ. Sydney), 69 Liverpool Road, Summer Hill, New South Wales.

Mugridge, William Alexander, M.B., B.S., 1956 (Univ. Sydney), 148 Hampden Road, Artarmon, New South Wales.

## Deaths.

The following deaths have been announced.

FAY.—Roscoe Woodrow Fay, on December 25, 1958, at Sydney.

HOLLAND.—John Joseph Holland, on January 4, 1959, at Perth.

## Medical Appointments.

Dr. J. A. B. Rolland has been appointed Honorary Surgeon in the Ear, Nose and Throat Department (with the status of Honorary Assistant) at the Queen Elizabeth Hospital, Adelaide.

Dr. G. F. Donald has been appointed Honorary Dermatologist (with the status of Honorary Assistant) at the Queen Elizabeth Hospital, Adelaide.

\* Annual donations.

Dr. A. A. Tye has been appointed Honorary Ophthalmologist (with the status of Honorary Assistant) at the Queen Elizabeth Hospital, Adelaide.

Dr. A. W. Burnell has been appointed Honorary Physician (with the status of Honorary Assistant Physician) at the Queen Elizabeth Hospital, Adelaide.

Dr. D. M. Williamson has been appointed Honorary Clinical Assistant at the Queen Elizabeth Hospital, Adelaide.

## Diary for the Month.

JAN. 19.—Victorian Branch, B.M.A.: Finance, House and Library Subcommittees.

JAN. 20.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

JAN. 22.—Victorian Branch, B.M.A.: Executive of the Branch Council.

JAN. 23.—Queensland Branch, B.M.A.: Council Meeting.

JAN. 27.—New South Wales Branch, B.M.A.: Medical Politics Committee.

JAN. 28.—Victorian Branch, B.M.A.: Branch Council.

## Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales. Anti-Tuberculosis Association of New South Wales. The Maitland Hospital.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

## Editorial Notices.

ALL articles submitted for publication in this Journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations, other than those normally used by the Journal, and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those of the list known as "World Medical Periodicals" (published by the World Medical Association). If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors submitting illustrations are asked, if possible, to provide the originals (not photographic copies) of line drawings, graphs and diagrams, and prints from the original negatives of photomicrographs. Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary is stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this Journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

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